Postoperative complications of thoracic surgery: Their recognition and treatment

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The author reviews the variety and incidence of complications following thoracic surgery and anesthesia; those most frequently encountered are most thoroughly discussed. Recognition and treatment of the most common respiratory and cardiac abnormalities are emphasized.

Studies have shown that thoracic patients are more prone to complications of surgery, particularly those related to the respiratory and cardiovascular systems, than are patients undergoing surgery at other sites.

Tarhan has documented a 29% incidence of myocardial reinfarction in patients undergoing thoracic surgery compared to an incidence of 8% reinfarction in patients undergoing upper abdominal surgery, and a 4% incidence in patients having surgery on other sites. The conclusions of this study are similar to those of other studies which have related an increase of pulmonary and other complications to thoracic surgery.

Since many patients scheduled for thoracic surgery have a history of pulmonary disease such as bronchitis, emphysema, or cardiovascular disease, and since the age range of the patients undergoing thoracic surgery may be higher than that of patients having other types of surgery, it is probable that events other than the actual surgery may influence the incidence of complications.

Many types of complications are noted following thoracic surgery. These include hypoventilation occurring as a result of analgesic medication, pain and/or splinting, atelectasis, pneumonia, pulmonary edema, adult respiratory distress syndrome, laryngeal edema or laryngotracheal erosions post-intubation. Cardiac complications include a higher incidence of atrial fibrillation post-lung resection, other arrhythmias, and an increased potential for myocardial infarction and congestive heart failure.

Catecholamine levels have been found to increase following lung surgery. This leads to an increased sympathetic effect on the vasculature as well as potentiating the probabilities of cardiac effects. It has also been noted that there is an increased incidence of fibrinolysis following lung surgery or surgery involving the pulmonary arteries. This may lead to increased postoperative bleeding.

In preparing an anesthesia care plan for patients with pulmonary or cardiac disease, the following preoperative tests are important to assist in determining the need for an enhanced level of postoperative respiratory management. The forced expiratory volume at one second (FEV₁) has been noted to be the most effective pulmonary function test to determine the adequacy of ventilatory capacity postoperatively. If a patient’s FEV₁ is under one liter, or 50% of the norm, a need for further preoperative study is indicated. This includes arterial blood gas studies indicating a PACO₂ under 50 or a PAO₂ over 60.

If these values are not present, the potential need for postoperative respiratory care and long-term ventilation is increased. If a patient has co-
existent restrictive or obstructive pulmonary disease, the incidence of postoperative respiratory inadequacy is also enhanced.

**Atelectasis**

A few of the more important and more common postoperative complications have been selected for further discussion. The first is atelectasis, defined as a collapse of a portion of the lung, usually due to bronchial obstruction. Alveolar ventilation is reduced in the affected area. In order to compensate for the reduction, alveolar ventilation is subsequently increased in the unaffected areas because of chemoreceptor stimulation. Carbon dioxide elimination may be adequate and, therefore, is not an accurate measure of the atelectasis present. The hydrogen ion concentration is also not predictable, since this may be dependent on carbon dioxide elimination. In this type of case, one might see a decrease in arterial oxygen without noting an increase in CO₂.

The causes of atelectasis frequently observed after surgery, particularly after thoracic surgery, include retained secretions, blocked airway passages from blood or dried secretions, airway closure, and altered surfactant. The symptoms noted are fever in the first 48 postoperative hours and tachycardia. It is possible that tachypnea will be present but this is not a consistent finding.

In the last five to 10 years, there has been discussion of airway closure as a leading cause of atelectasis. If the critical lung volume is larger than the functional residual capacity of the lung, the airways close. The small airways, those leading to the alveoli which are under one mm in diameter, show a tendency to close during exhalation as the lung volume decreases. In order to prevent closure, the critical lung volume must be exceeded by the functional residual capacity.

Factors which increase the potential of airway closure include: the supine position, restrictive lung disease, pain induced splinting, respiratory depression from narcotics or relaxants, and abdominal distention. Many of these factors are likely to occur after thoracic surgery, increasing the probability of limiting lung expansion. If airway closure occurs it becomes difficult, proportionate to the length of time of closure, to expand the affected alveoli.

In order to prevent or treat atelectasis, one must realize the importance of encouraging the patient to turn, cough, and deep breathe at least every two hours. Deep breathing should include a slow inspiration with breath holding at the peak of inspiration. If it is difficult to encourage the patient to cough, finger pressure on the trachea in the area of the supra-sternal notch will cause involuntary coughing.

Methods of preventing atelectasis should be discussed with the patient preoperatively, when he is psychologically more attuned to learning about his postoperative state and better able to understand the importance and reasons for the postoperative requirements. He must also be encouraged postoperatively to make every effort to do the indicated exercises correctly.

The patient should be ambulated as soon as possible so that bronchial secretions may be mobilized and expelled. Respiratory therapy assists in the mobilization and expulsion of secretions as does chest physiotherapy and postural drainage. These techniques should be utilized approximately 45 minutes to one hour after the administration of a pain medication as the procedure may be painful to the thoracic patient and that may limit the quality of participation he is able to achieve.

The incentive spirometer may be incorporated in postoperative care to emphasize large volume inhalation and give the patient an indication of his effort as reward. Emphasis on inspiration is necessary to decrease the potential for airway closure. Potential for closure may be enhanced with techniques emphasizing exhalation.

In the past, blow bottles were used frequently following thoracic surgery to decrease atelectasis. This technique does emphasize exhalation and may increase the potential for airway closure. For practical purposes their present use is almost nonexistent.

The ultrasonic nebulizer may be used to hydrate secretions; however, it may also cause irritation leading to bronchospasm. In addition, fluid from the nebulizer may be readily absorbed leading to fluid and electrolyte imbalance.

Nasotracheal suction and bronchoscopy may relieve obstructive secretions if other methods are not adequate. It is possible however, that neither of these methods will locate a definite area of obstruction and more general methods of relieving atelectasis must again be utilized.

There has been one study indicating the possibility of utilizing bronchial blockers to assist in giving positive pressure ventilation to expand atelectatic areas. This, however, has not been accepted at this time as a universal method for relieving atelectasis.

Other common respiratory therapy measures used to increase functional residual capacity include positive end-expiratory pressure (PEEP) and intermittent positive pressure breathing (IPPB). If
IPPB is given, it is necessary that the volume administered exceed the volume that the patient may spontaneously generate. If this is assured, the alveoli are expanded, shunt is decreased, and the arterial oxygen may be increased. For most effective IPPB, the patient must be taught preoperatively how to utilize the equipment and an experienced therapist must be watching every breath, ensuring that the patient and the machine generate an adequate volume. IPPB may also deliver humidified medication such as bronchodilators or expectorants.

In the last few years Continuous Positive Airway Pressure (CPAP) and Intermittent Mandatory Ventilation (IMV) have been utilized as methods of preventing postoperative airway closure and atelectasis. CPAP utilizes spontaneous ventilation on the part of the patient while exhalation is done against a water column of the desired pressure resistance. This decreases airway closure. In order to avoid rebreathing from the long tubing utilized in this technique, the gas flow must be two to three times the patient’s minute ventilation.

IMV utilizes spontaneous ventilation from a fresh gas source of low resistance. It may be used with or without positive end expiratory pressure. Intermittently, at pre-set intervals, the patient receives active inflations from the ventilator. This assists in keeping alveoli open when the patient may not be able to generate adequate volume for a sigh with his spontaneous breathing pattern.

When setting a ventilator for the postoperative thoracic patient, one should utilize a tidal volume of approximately 12-15 cc/kg and a rate of 10-12/min. At present it is felt that it is probably best not to utilize a sigh in the ventilator pattern; however, this is still a topic of some debate. The inhalation/exhalation ratio should be approximately 1:2. If the patient requires more time for inhalation, the ratio should never fall below 1:1. If it does, the rate should be changed to allow for increased exhalation time so that air is not trapped in the small airways and alveoli. Trapping does not permit adequate exchange at the alveolar-membrane level.

Hypoventilation and hypoxemia

Hypoventilation and subsequent hypoxemia may occur commonly in the postoperative thoracic patient. The definition of hypoventilation is inadequate alveolar ventilation of such magnitude that the oxygen tension and saturation rapidly decline. Hypoxemia is defined as a PaO₂ under 60 mm of mercury. Causes include postoperative pain, analgesic medications, atelectasis, pneumonia and pulmonary embolus. The symptoms noted include tachycardia, tachypnea, disorientation, hypotension and, if the PaO₂ falls below 40, cyanosis, bradycardia and hypercapnea.

Usually, the PaCO₂ is low (because of the tachypnea) until depression is such that the respiratory rate falls. The PaO₂ generally must be under 40 mm of mercury to depress the respiratory center so that the respiratory rate falls and hypercarbia develops. Treatment of this phenomenon is intubation and ventilation. It may be prevented by avoiding the desire to extubate early following surgery.

In determining criteria for extubation and discontinuing mechanical ventilation, the following principles may be helpful:

1. The vital capacity should be twice the resting preoperative tidal volume before extubation is considered if lung parenchyma has not been removed. This, of course, will be changed following lung resection. Two-thirds of the expected level might be used to determine adequacy.

2. Inspiratory capacity should be under −10 cm of water. The inspiratory capacity is a more useful measure than the expiratory capacity as it helps to maintain open airways by placing maximum patient effort on inspiration. It also appears to be more indicative of adequate lung function. If the expiratory capacity is used as a measure, this should be over +20 cm of water pressure.

3. It should be possible to maintain arterial oxygen saturation above 95% without ventilation prior to extubation. Extubation should not be rushed since the vital capacity normally decreases postoperatively because of pain, narcotics, or removal of lung parenchyma. Ciliary action may be decreased by the use of analgesics, anesthetics, and atropine, and the cough reflex is decreased by anesthesia and narcotics. These effects decrease the patient’s capacity to rid himself of secretions.

4. One hundred percent oxygen should be avoided in the postoperative period if possible to prevent reabsorption atelectasis. If the patient has been on 100% oxygen, it is possible that a large percentage of oxygen has been taken into the blood, leaving an inadequate alveolar volume to prevent alveolar and airway closure. It is recommended that a cushion of nitrogen be present as it is in room air so that reabsorption of oxygen does not lead to alveolar collapse.

If prolonged intubation is needed (24-48 hours or longer) a nasotracheal tube may be substituted for the orotracheal tube utilized during anesthesia. This will minimize patient struggling and incompatibility with the ventilator. The ad-
Equacy of ventilation should be monitored at frequent intervals. The ventilator should be checked, the tidal volume, inspiratory rate, and expiratory time should be monitored, and arterial blood gases adjusted according to the parameters monitored. It is also important to monitor hemodynamic performance as continuous positive pressure ventilation may decrease the venous return to the heart and cardiac output.

During mechanical ventilation, the patient often has a lower urine output and decreased renal function. This is partially due to stimulation of ADH by mechanical ventilation, narcotics, and anesthetic agents. Blood flow may be redistributed with positive end expiratory pressure and renal blood flow may be decreased further diminishing output. If the patient is on diuretics postoperatively and the osmolality of the blood increases above 320, the risks of renal failure increase. If renal failure ensues there is an increase in the mortality of respiratory failure in the postoperative period.

Parenteral nutrition is very important to maintain adequate fluid and electrolyte balance while the patient is on mechanical ventilation but the composition of this nutrition is often the source of water, acid-base, or metabolic derangement. The type of fluids given should be an important consideration. Intravenous dextrose, amino acids, vitamins, and lipids should be considered early for patients on long term ventilation. This will help maintain fluid, electrolyte, and metabolic balance and will therefore assist in the earliest possible termination of ventilation.

To ensure adequate postoperative ventilation, the oxygen saturation should be kept above 95% and the carbon dioxide content of the blood should be between 35 and 40 torr. If this is not the case, one should check for systemic infection, agitation, pain, shivering or other causes of metabolic derangement. If the patient needs treatment for synchronization with the ventilator postoperatively, one should first try to determine the cause of respiratory failure and change the ventilatory pattern to better suit the patient’s needs. The patient may be removed from the ventilator and hyperventilated by hand to determine the ease with which the patient adapts to ventilation with a lowered PACO₂. The patient should adapt easily to ventilation with a PACO₂ of 35-40 and fall asleep if there is no other cause of insufficiency.

If metabolic acidosis is present it may be treated. Shivering may be treated with Thorazine® in 2 mg IV boluses, increasing the amount until the shivering stops. Shivering may also be treated with muscle relaxants. Thorazine® is perhaps the treatment of choice in those patients who are able to maintain cardiovascular stability. Thorazine® does have a mild alpha blocking effect, however, which dilates blood vessels and increases the need for volume in patients who are volume depleted postoperatively.

If the cardiovascular and fluid balance status have not been determined prior to the administration of Thorazine®, hypotension could result. The half life of Thorazine® is approximately 8 to 12 hours; it is quite possible that the hypotensive effects may last longer than desired, leading to difficulty in maintaining adequate tissue perfusion. In the case where the patient is unable to tolerate Thorazine®, the use of a long acting muscle relaxant, such as pancuronium, to achieve paralysis may be indicated. If this is done the patient should be sedated so that he is unaware of his inability to move.

Morphine may be given in 2 mg boluses until an adequate level of sedation is reached. Most patients after thoracic surgery will require only 2-6 mg of morphine for sedation, adequate to enable them to tolerate ventilation. Patients who are younger or more agitated may take up to 30 mg of morphine every 3 to 4 hours in order to tolerate mechanical ventilation. If relaxation is an adjunct in producing toleration of ventilation, it is possible that lower doses of morphine may be effective in even the most agitated patients.

The importance of maintaining adequate sedation while using neuromuscular blocking agents should be emphasized, since these agents do not block the sympathetic effects of agitation and apprehension which may produce unwanted results on the cardiovascular and respiratory systems. Any patient who has central nervous system dysfunction and is being ventilated on a long-term basis after thoracic surgery should probably be maintained with ventilation by relaxants alone so that his central nervous system function can be better evaluated. This is perhaps one of the only situations where relaxants should be used without sedation.

**Pain relief**

Pain relief has been mentioned as a potential cause of ventilatory inadequacy. Since thoracotomy is one of the most painful surgical operations for the patient, relief of this pain in the postoperative period is important in any discussion of postoperative care. The methods most frequently used for pain relief are analgesics, intercostal block (either continuous or given in a single injection during surgery), or continuous epidural. The latter two
methods may be used in combination to decrease the need for analgesic agents.

Regional block was first described for thoracotomy patients in the 1940's. This technique has recently received a resurgence in popularity and has become more frequently used in the treatment of postoperative thoracic pain. Afferent pain impulses are blocked by this method, yet the cough reflex is preserved. When narcotic analgesics are used for anesthesia, the cough reflex is depressed.

Regional block permits maintenance of adequate vital capacity while relieving discomfort, thus decreasing mucous collection and atelectasis which might be seen with either no analgesic or with analgesics in adequate doses to prevent the pain but depress ventilation. If given in combination with narcotics, the block decreases the need for these drugs so that ventilatory adequacy is preserved.

A study of Bergh, Dottori, et al. regarding the effect of intercostal block on lung function post-thoracotomy indicated that intercostal block supplemented by low doses of analgesics maintained a better ventilation/perfusion ratio and allowed earlier postoperative restoration of the vital capacity than could be achieved with narcotic analgesics alone. According to this study, the best agent to be used for an intercostal block is Marcaine® with epinephrine. This agent had a block duration of approximately 10.4 ± 2.2 hours, while the shorter acting agents such as Carbocaine® mixed with Xylocaine® had a block duration of 4.7 ± 0.7 hours.

The effects gained for the patient given Marcaine® included no perception of the initial, more severe pain common during the immediate postoperative period, and with only small doses of narcotic analgesia, a significant increase in pulmonary function over the patients given shorter acting regional agents such as Carbocaine® combined with lidocaine. There was also increased pulmonary function over the group of patients given narcotic analgesics alone.

Between the fourth and sixth day, the amount of analgesics given to all three groups became equal and at the tenth day, there was no significant difference in oxygen saturation between the Marcaine® group and the analgesics-only group. This study indicates that Marcaine®, a long acting nerve blocking agent given as an intercostal block, is a significant adjunct in decreasing post-thoracotomy pain and in increasing pulmonary function for the post-thoracotomy patient.

Arrhythmias

In addition to pulmonary function changes in the postoperative period, arrhythmias and changes in cardiac function are very frequent complications. Post-thoracotomy, 9 to 21% of the patients have been noted to have arrhythmias. Causes of these arrhythmias include: (1) intra or postoperative hypotension with subsequent cardiac hypoxia; (2) increased volume load related to a decreased vascular bed caused by removal of lung parenchyma; (3) pain; (4) acid base or electrolyte imbalance; (5) vagal and sympathetic stimulation by suction or other cause; (6) pulmonary embolus; and (7) myocardial infarction.

The FEV as a postoperative study was determined to have no correlation with the occurrence of arrhythmias. Arrhythmias frequently seen post-thoracotomy include the following.

Sinus tachycardia is a frequent arrhythmia following thoracic surgery. It is defined as a sinus rate of 102-150 beats/min. A rate of up to 120 beats/min is expected after thoracic surgery due to sympathetic stimulation and pain. Treatment of this arrhythmia includes treatment of pain, infection, fever, shock, and maintenance of adequate ventilation and blood volumes which may be decreased from hemorrhage. Edrophonium may be used as a longer acting agent if needed. The cause of tachycardia should be treated prior to using an agent to treat the arrhythmia itself.

Atrial fibrillation is the most common arrhythmia noted after thoracic surgery, and is defined by an irregular rate of 150 beats/min or more. The cause is probably due to acute atrial dilatation because of the removal of lung parenchyma. There is a smaller pulmonary bed following surgery which must accommodate the same fluid volume; therefore, more blood is returned to the heart. The cardiac work is increased to pump blood through a more restricted vascular bed.

The incidence of atrial fibrillation or possible congestive heart failure after pulmonary resection depends upon the underlying cardiac reserve and is increased in patients who may have a small cardiac reserve prior to surgery. The incidence also appears to be related to the cause of resection, according to a study by Beck-Nielsen et al. If resection is done for malignant disease, the incidence of atrial fibrillation is approximately 19.6%. If resection is done for benign disease, there is a reported incidence of 3.1%.

The type of surgery eliciting atrial fibrillation in the postoperative period is related most commonly to patients undergoing the most extensive surgery. Patients who had pneumonectomy had an
incidence of 22.2% atrial fibrillation. Those having lobectomy had an incidence of 20.3%, and those having exploratory thoractomy had an incidence of 16% atrial fibrillation. These differences in incidence may be due not only to the fact that the disease is malignant or benign, but also to the fact that malignant disease usually requires more extensive surgery and is often done on an older age group of patients. Patients requiring resection for malignant lesions also often have a history of smoking with the possibility of bronchitis and emphysema.\textsuperscript{11}

Treatment of atrial fibrillation includes the possibility of using prophylactic digitalization or quinidine preoperatively. Cardiologists have used this method of treatment in order to prevent atrial dilatation and arrhythmias after pulmonary resection. Beck-Nielsen et al felt, however, that this preoperative prophylactic treatment was not indicated on all thoracic patients. They treated occurrence of atrial fibrillation with: (1) DC cardioversion at 25-50 watt seconds; (2) DC cardioversion at 2.5-5 watt seconds increasing until successful for patients on digitalis; and (3) digoxin at 0.75 mg IV postatrial fibrillation followed by 0.25 mg every 8 hours for a total of 1.5 mg. The maintenance of digitalis may then be kept at .25 mg every day.

If the patient is on digitalis prior to cardioversion, the electrical stimulus will increase the chance of digitalis toxicity.

It is therefore indicated that attempts to cardiovert should start at a lower intensity stimulus. For the same reason, digitalis should be held until after cardioversion for those patients not previously on this drug. Digitalis can assist in maintaining a slower heart rate to decrease the potential for recurrence of atrial fibrillation. In the study by Beck-Nielsen, approximately one-third of the cases had atrial fibrillation lasting only 24 hours with treatment and most others lasted only one to three days. All had reverted to normal rhythm by the time of discharge.\textsuperscript{11}

Paroxysmal supra-ventricular tachycardia is an arrhythmia caused by an increased atrial load or sympathetic stimulation postoperatively. Treatment consists of: (1) carotid sinus massage, (2) cardioversion, particularly if the patient is prone to congestive heart failure, (3) Tensilon\textsuperscript{\textregistered}, (4) digitalis, (5) propranolol, and (6) digitalis/propranolol combination.

The digitalis/propranolol combination is recommended by many internists as the propranolol tends to decrease the inotropic action of the heart while the digitalis tends to maintain this. Thus, they may supplement each other for better control of cardiac activity.

Ventricular tachyarrhythmias and PVC's. The causes of these arrhythmias include: (1) electrolyte abnormalities, (2) suction, (3) vagal stimulation, (4) acidosis, (5) hypoxia, (6) hypovolemia, (7) shock, (8) pain, (9) fever, and (10) digitalis intoxication. Since all these causes may be observed frequently after thoracic surgery, ventricular arrhythmias may be common.

Treatment of ventricular arrhythmias may be instituted by utilizing the following drugs:

1. Pronestyl\textsuperscript{\textregistered}—250-500 mg every 4 to 6 hours IM.
2. Quinidine—200-300 mg every 6 hours if Pronestyl\textsuperscript{\textregistered} does not help, or in the case of digitalis intoxication.
3. Dilantin\textsuperscript{\textregistered}—100 mg four times every day.
4. Lidocaine—1 mg/kg followed by a drip of 1-3 mg/min.
5. Propranolol—025-10 mg until the desired treatment is obtained. If propranolol is used, one must realize that it has a negative inotropic effect on the heart with the potential for precipitating congestive heart failure in patients prone to this problem.

Ventricular tachycardia. The causes of this arrhythmia after thoracic surgery may be: (1) digitalis intoxication, (2) hypoxia, and (3) increased cardiac activity required after decreasing the pulmonary vascular bed by removal of lung parenchyma.

Treatment consists of: (1) a bolus of 1 mg/kg of lidocaine followed by a lidocaine drip, and (2) cardioversion with a DC shock of 250-400 watt seconds.

Bradyarrhythmias may be seen postoperatively due to: (1) digitalis intoxication, (2) excess vagal tone, (3) decreased liver function, and (4) jaundice and subsequent increase in metabolic products. Treatment consists of (1) atropine, (2) withholding digitalis, and (3) use of a pacemaker, if the arrhythmia is due to block.

If atropine does not treat the bradyarrhythmia and the cardiac output is so low that tissue hypoxia is eminent, small doses of isoproterenol given in a drip may be effective in increasing the heart rate until a pacemaker is inserted. This drip should be carefully titrated to give only an adequate heart rate. The drug itself may cause tachycardia, arrhythmias and increased cardiac activity. This may cause subsequent myocardial hypoxia if the heart rate is high and coronary artery filling time decreased.
Myocardial Infarction

The incidence of myocardial infarction, according to the study by Tarhan, is increased post-thoracotomy. He reports that of 93 patients having a previous infarction and undergoing thoracic surgery, 89% had a reinfarction postoperatively. Out of 84 patients having upper abdominal surgery, 8% had reinfarction, and out of 410 patients having surgery on other sites, 4% had infarction.12

The symptoms of myocardial infarction may be disguised after thoracotomy since pain may not be an accurate guideline. The thoracotomy itself causes pain which may not be distinguishable from pain of infarction. The only symptoms noted may be tachypnea, tachycardia, hypotension, and arrhythmias. On the ECG one should look for enlarged Q-waves rather than looking mainly for ST-T wave changes as one would in myocardial infarction without surgery. ST-T wave changes may also be related to pericarditis, drug therapy electrolyte changes or autonomic imbalance, thus leading to confusion of diagnosis.

If the infarction is large enough and the cardiac reserve is limited, congestive heart failure may develop, particularly after lung resection because of a decreased vascular bed and decreased ability for blood oxygenation. The incidence of myocardial infarction and reinfarction may be exaggerated after surgery due to the increased cardiac work required by pain and other sympathetic stimulation, decreased area of lung parenchyma, hypoxia, hypoperfusion, fever, anemia or changes in blood volume from IV therapy.

Delius, Johansson, Nystrom, et al found that catecholamine output is increased postoperatively in the thoracotomy patient. This could cause an increase in cardiac work and hypertension, leading to an increased need for myocardial oxygenation but providing a decreased coronary filling time. Delius found that adrenalin output increased 400% during the first five postoperative hours. It then decreases and nor-adrenalin output increases. The adrenalin output seemed to correlate with the maximum temperature rise. The increase was not correlated with the type of lung surgery, hypoglycemia, hypoxia, acidosis or hypertension.13

Since increased sympathetic activity increases the work load and oxygen requirements of the heart, everything possible should be done to limit the need for increased oxygenation of the myocardium. This may entail close monitoring of the patient, ventilation and sedation with a slow emergence.

As one may note from the multiple aspects influencing the postoperative outcome of the patient following thoracic surgery, it is imperative that we realize that other factors may be utilized in addition to the anesthetic level to determine readiness for extubation and removal from an intensive care situation, either in the recovery room or the intensive care unit.

Conclusion

This brief resume of a complex problem has attempted to delineate a few of the difficulties which may be important to the care of the post-thoracotomy patient. These are by no means the only difficulties which could be encountered, but the most frequent difficulties have been noted. It is important to realize the increased potential for the development of problems in the thoracotomy patient and the importance of good preoperative teaching so that the best ventilatory capacity possible may be maintained after surgery.

Monitoring should be adequate to the patient's needs and postoperative ventilation maintained until the patient is able to adequately ventilate himself without increasing the work of breathing and subsequently causing added strain on the heart. It has been noted that without adequate care, the work of breathing can increase from 1% of the basal metabolic rate to 10%, thus expending the patient's energy which could be better utilized in other aspects of recovery. The weaning process should be done according to the patient's capability to maintain his own homeostasis.

In the thoracotomy patient, the anesthetist's work does not stop when the case is completed, however. The patient's progress should be followed and anesthesia input maintained until he is able to function without ventilatory or cardiac assistance. Observant care by anesthesia personnel is imperative to successful postoperative care.

REFERENCES


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