

PULMONARY PHYSIOLOGY ALTERATIONS FOLLOWING CARDIAC SURGERY

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Cardiac surgery has significant and well-studied effects on pulmonary physiology. A major impact of surgery is to decrease pulmonary compliance (C_p) (1,2,3). Causes for the decreased C_p include increased pulmonary capillary volume and extravascular lung water because of the crystalloid solution patients receive in the course of cardiopulmonary bypass (CPB), as well as microatelectasis that is multifactorial in etiology (1). Atelectasis is caused, in part, by anesthetic and neuromuscular blockade (paralysis), which typically cause cephalad shift of the diaphragm in supine patients. This decreases functional residual capacity (FRC) below the lungs' closing volume (4). The lungs are also typically deflated to some degree during cardiac surgery to allow clear access to the to the heart, even if the procedure is done off CPB and through new "minimal access" techniques. This, as well, contributes to atelectasis. Chest wall compliance, by view of recent studies (5), is also decreased after sternotomy, though minimally and for only a brief (<4 hours) period. Airway resistance (AR) is generally increased after cardiac surgery (2) for unclear reasons, but possibly because of bronchial edema.

The clinical effects of these physiologic changes are primarily two. Ventilation/perfusion (V/Q) relationships are altered creating physiologic shunting through poorly ventilated lung units resulting in an increased alveolar-arterial oxygen difference (A-a O_2D). Though this effect can be very different between patients, it is on average about 70 mm Hg. To prevent hypoxemia, therefore, post cardiac surgery patients require, typically, increased fraction of inspired O_2 (FiO_2). It is noteworthy that this effect is seen in patients whose surgery is performed off CPB, through minimal access approaches, in addition to patients receiving traditional treatment (6). This effect can last only hours or for a few days, depending upon the patient. The other effect of physiologic changes post surgery is that patients experience increased work of breathing (WOB) consequent to the increased AR, and decreased C_p . For the vast majority of heart surgery patients this is inconsequential to their clinical course. However, in patients with significant underlying neuromuscular disease or pulmonary pathology, especially those with chronic obstructive lung disease (COPD), these changes may prove troublesome and result in prolonged mechanical ventilation (MV).

INCIDENCE OF PULMONARY COMPLICATIONS

There are many potential pulmonary complications of cardiac surgery. They will be individually discussed in detail through the other sections of this chapter. However, it is helpful to know the general incidence of the more common and morbid problems.

- Prolonged MV (>48 hours), is required in from 10 - 23% of post cardiac surgery patients (7,8).
- Atelectasis of a great enough extent to be present on chest X - ray (CXR), is common post cardiac surgery. One recent study reported an incidence of left lung atelectasis at 88%, and right lung Atelectasis at 61% (9).
- Pulmonary edema, to greater or lesser degrees, typically is present in patients post CPB (10).
- Diaphragm dysfunction, not necessarily effecting clinical outcome, but when looked for aggressively with sophisticated techniques, is present in 25% to 50% of post cardiac surgery patients (11).
- Pleural effusions are common after cardiac surgery,

occurring in 40% to 90% of patients depending upon the series (12,13). Most effusions are small in size, however, and do not require treatment. Large effusions (>25% of the hemithorax) occur in slightly less than 1% of cardiac surgery patients (14).

- The Adult Respiratory Distress Syndrome (ARDS) occurs in 1% to 2% of post cardiac surgery cases but is associated with significant mortality (15).
- Pneumonia occurs in 4 - 6% of cardiac surgery patients (16,17).
- Pulmonary embolism (PE) is thought to be rare after cardiac surgery, but in a recent large series it complicated 3% of the cases (18). When PE occurs post cardiac surgery, it is associated with a mortality of 18% to 34% (18,19).
- Pulmonary arterial hypertension is common after cardiac surgery, but is only unusually associated with notable clinical problems. However, patients with severe biventricular failure and those undergoing congenital heart surgery, mitral valve replacement, orthotopic heart transplant, and left ventricular assist device placement are at particular risk for the development of severe pulmonary hypertension which can lead to right ventricular decompensation and, rarely, mortality (20).

PREOPERATIVE EVALUATION AND PULMONARY COMPLICATION PREVENTION

The risk factors for pulmonary complications following cardiac surgery can be divided into specific pulmonary pathologies, usually present long before the need for surgery, and other “nonpulmonary” conditions, which can result in prolonged MV. The following are the major lung conditions, listed in order of importance, that can dispose the post heart surgery patient to even greater than expected impairment of oxygenation, prolonged MV, atelectasis, and pulmonary infections (4,21):

- COPD characterized by pulmonary function tests (PFTs) showing a forced expiratory volume in one second (FEV- 1) of <1.5 liters (L) or an FEV - 1/Forced vital capacity (FVC) ratio of <65%. This is especially so for patients with the chronic bronchitis variant of this disorder with its attendant

large volume of sputum production, and for patients with chronic hypercarbia ($CO_2 >45$).

- Asthma, poorly controlled.
- Active pulmonary infections
- Restrictive lung diseases such as idiopathic pulmonary fibrosis (IPF).
- Smoking history of generally greater than 20 pack years.
- Obesity

One early study (22) reported a postoperative incidence of pulmonary complications in patients with abnormal preoperative PFTs to be 42%. With optimization of pulmonary function in these “at risk” patients before and after surgery, usually by treating COPD with bronchodilators and pulmonary secretion management techniques, the complication rate was cut by two thirds. More recent studies have not shown such dramatic improvement in complication rates of “at risk” patients by aggressive, targeted perioperative care (23). It appears, though, that these patients are now routinely receiving some pre and postoperative pulmonary therapy, due to increased awareness of pulmonary complications, making it difficult to demonstrate major improvements in studies. We recommend, therefore, screening patients for these risk factors, and optimizing the pulmonary status of patients who have them pre and post surgery. In addition to the risk factors identified above, we also recommend for pulmonary evaluation those patients with dyspnea out of proportion to their cardiac pathology. The evaluation should include, at least, PFTs and a plain chest radiograph. There are no PFTs that are absolutely prohibitive for cardiac surgery (24). The choice of whether or not patients with lung disease have cardiac surgery is an individual one which entails measuring the risks versus benefits for the individual patient.

Therapy for patients with COPD includes pre and postoperative inhaled bronchodilators (see *Drug Table 1* at the end of the chapter) routinely. If the patient has had a recent exacerbation of COPD, we will often treat with a short course of steroids preoperatively to optimize lung function. Chronic bronchitis will require special attention to pulmonary secretion clearance postoperatively. This includes aggressive endotracheal suctioning of patients on MV in the recovery room (RR), for the immediate postoperative period. A closed suctioning system that attaches to the endotracheal tube (ETT) is more convenient for the nursing and respiratory therapy staff and often, therefore, results in more frequent suctioning. However, if

secretions are thick and difficult to remove, traditional “open” (patient disconnected from the ventilator) ETT suctioning with sterile saline and bagging is more effective. Mucolytics are often employed in either inhaled form (acetylcysteine) or enterally (guifenesin) (see *Drug Table 3* at the end of the chapter). It should be born in mind that inhaled acetylcysteine can sometime provoke bronchospasm. If asthma is not optimally controlled preoperatively with the usual chronic agents, we will treat with a short course of steroids. Pulmonary infections should be treated with appropriate antibiotics preoperatively, and completely cleared before proceeding to surgery Patients with interstitial lung disease can be expected to require higher levels of FiO₂ for longer periods of time post operatively. This group of patients is certainly at greater risk of developing additional pulmonary complications, but is at notably less risk than patients with obstructive lung disease (24). Smokers should stop tobacco use prior to surgery. Extrapolating from the general surgery literature, patients should be smoke free for at least six weeks prior to surgery for maximal benefit (25). Obesity has traditionally been considered a major risk factor for pulmonary complications after cardiac surgery. Although obesity clearly increases the A-a O₂D post cardiac surgery to a mild or moderate degree (26), and slightly increases the risk of postoperative PE (18), recent large series have failed to confirm it’s association with other major pulmonary complications such as prolonged mechanical ventilation (27,28). Therefore, obese patients should be encouraged to lose weight prior to cardiac surgery, but it does not appear to be critical in preventing postoperative pulmonary complications.

Typically, all patients going for cardiac surgery are taught, before surgery, the benefits, and techniques of coughing and deep breathing post operatively. This involves demonstrating the use of pillows specifically made for sternotomy patients to brace the surgical site in the several days post operatively, as well as the use of the incentive spirometer, which we recommend hourly in our typical post operative patients.

Nonpulmonary conditions associated with prolonged MV after cardiac surgery according to recent large clinical series (29,30), include (with relative odds ratios):

- Age >65 years (1.31).
- Inpatient hospitalization before surgery (1.39)
- Peripheral vascular disease (1.26)

- Cardiogenic shock (2.54) or severe left ventricular dysfunction (1.27)
- Renal insufficiency (1.27)
- Serum albumin of <4.0 g/L (1.24)
- Systemic oxygen delivery of <320 mL/min/m² (1.27)
- A “redo”operation, reexploration of the chest for bleeding, or delayed sternal closure (1.47)
- procedures involving the thoracic aorta (1.92)
- transfusion of greater than 10 units of blood products (1.87)
- CPB time of greater than 120 minutes (1.23)

These conditions promote prolonged MV through a variety of mechanisms and do not generally lend themselves to preoperative remediation. It is helpful, however, to understand their impact on the post CS pulmonary course.

MECHANICAL VENTILATOR SUPPORT IN THE IMMEDIATE POSTOPERATIVE PERIOD

Following cardiac surgery, patients are taken off CPB and sent back to the RR on a mechanical ventilator. Uncomplicated patients will require mechanical ventilatory support typically for some hours until they are ready to “wean” from the ventilator and have their ETT tube removed. During this time, the patient recovers from hypothermia, anesthesia, neuromuscular blockade, and typically regains hemodynamic stability. The ventilator for this period is set in a “support mode”, that is a mode, which essentially does all of the WOB for the patient. The two commonly used support modes are assist control (AC) and synchronized intermittent mandatory ventilation (SIMV) with pressure support (PS). These are “volume cycled ventilator modes”. In each of these modes a respiratory rate and tidal volume is set for the ventilator. For example, a typical respiratory rate (RR) would be 10 and tidal volume (Vt) 800 ml. In both of these modes the patient will, at least 10 times a minute, get a Vt of 800 ml. In either mode, patients can initiate breaths between the preset Vts. In both modes the ventilator will augment those breaths, but in different ways. In AC, when the ventilator senses that the patient is initiating a breath, it will give the patient the preset Vt. For example, if our patient on AC set at a RR of 10 with a Vt of 800 ml

initiates 5 extra breaths a minute, each of those breaths will be 800 ml. The patient's total minute ventilation (V_e) will be 15 breaths x 0.8 L = 12 L. In the SIMV mode with PS, a PS level is selected in addition to a RR and V_t . When the patient initiates a breath between the preset V_t s, the ventilator will generate air flow through its inspiratory loop. The amount of airflow will be enough to maintain a positive airway pressure, at the selected PS level, while the patient is actively inspiring. The purpose of PS is to aid the patient in breathing through the ventilator tubing and circuitry. The size of the V_t that the patient actually receives depends upon how strongly he or she inspires, the level of pressure support, and the length of time that he or she inspires. For example, if our patient is placed on SIMV at a RR of 8 with a V_t of 800 ml and PS of 15 cm H₂O, V_e will be at least 8 x 0.8 L = 0.64 L. If, however, he initiates 5 extra breathes per minute which are pressure supported to a V_t of, on average, 0.5 L, his V_e will be 5 x 0.5 L = 0.25L added to the 0.64 L for a total V_e of 0.89 L. There are no studies to show which of these two volume-cycled-ventilator modes are superior in supporting patients post CS or supporting patients with respiratory failure in general. It is very important, however, that when the SIMV mode is used that it be used in conjunction with pressure support. Pressure support compensates for the work of breathing through ventilator circuitry. Without PS, the work of breathing through the ventilator can be fatiguing for patients' respiratory muscles, and actually prolong the need for MV (31). Typical settings for using the either the AC or SIMV ventilator modes are:

- RR: 8 - 12 breaths per minute
- V_t : 10 - 15 ml per kg
- PS: 5 - 15 cm H₂O (our institution routinely uses 10 in our post cardiac surgery patients)

These settings were developed from anesthesia experience to prevent both over distention of the lung resulting in "barotrauma" and under distention of the lungs resulting in atelectasis. These settings, additionally, will generally result in eucapnia, but it's appropriate and routine practice to check arterial blood gasses approximately 20 minutes after arriving in the recovery room. When patients arrive in the RR the FiO_2 delivered by the ventilator is typically 0.60 or 60%, as long as the oxygen carrying capacity of the blood is not compromised by an O₂ saturation of less than 90%. FiO_2 above 0.60 is generally considered to be toxic to the lungs through generation of oxygen radicals. Ventilators in common use typically offer several

options for the inspiratory flow pattern and allow peak inspiratory flow rate to be set. The "decelerating ramp" flow pattern is generally considered to deliver gas in the most physiologic way and is usually preferred. Peak inspiratory flow rates are generally initially set at 60 L/min but may be varied up and down substantially depending upon the needs of the patient. The latest generation of ventilators, utilizing sophisticated computer programs, will calculate the AR and C_p of the patient's lung, on a breath-to-breath basis, and deliver V_t s through a flow rate and pattern that is most physiologically appropriate. Positive end expiratory pressure (PEEP) use, as a standard post cardiac surgery, varies from institution to institution. Argument for using a low level of PEEP routinely in post cardiac patients is that it helps prevent microatelectasis. "Physiologic PEEP" is usually set at about 5 cm H₂O. Argument against its routine use is that it can negatively impact hemodynamics by increasing intrathoracic pressure, thereby decreasing venous return to the right heart as well as increasing right ventricular afterload. Again, no data exists as to whether routine use of PEEP in post CS patients is beneficial or not. At our institution, because of concern for hemodynamics we do not routinely use PEEP.

VENTILATOR ADJUSTMENT AND TROUBLE SHOOTING

The ventilator is adjusted in the early postoperative period based on arterial blood gas (ABG) sampling and "peak inspiratory pressure" (PIP). In general, the partial pressure of arterial CO₂ ($paCO_2$) is targeted to be 35 - 45 mm Hg, which, unless there is a concurrent metabolic acid base disturbance, should result in an acceptable pH of 7.35 - 7.45. $paCO_2$ is directly proportional to the alveolar ventilation (V_a) which is equal to the V_e minus the dead space ventilation (V_d) (that portion of the lung which ventilated but not perfused such as the airways). V_e is equal to the RR x the V_t . In normal lungs about 150 ml. of each V_t is V_d . V_a is, therefore, equal to the RR x ($V_t - 0.15$). So to treat respiratory acidosis or alkalosis that has developed on the ventilator the V_a is adjusted up or down in proportion to the amount of change in the CO₂ required to achieve eucapnea. This can be done by adjusting the V_t or the RR, which is usually preferred. Suppose for example, if we have a patient with a $paCO_2$ of 50 and a pH of 7.32 immediately post operatively on MV settings of AC, RR 8, and V_t of 800 ml who is not initiating any breaths. The V_a is equal to 8 x (0.8 - 0.15) = 5.2 L. To bring the pCO_2 down 20% to 40 mm Hg, the V_a

needs to be increased 20% to about 6.24 L. By increasing the RR to 10 the V_a will increase to 6.5 L, approximately the V_a needed. Partial pressure of arterial oxygen (PaO_2) is targeted to be greater than 60 mm Hg (correlating to an arterial oxygen saturation greater than 90%) hopefully using a FiO_2 of 60% or less. Ventilator strategies to improve oxygenation are discussed in the section on hyperemia.

It is important to pay attention to PIPs. Ventilators measure pressures in the ventilator circuitry as a way to estimate the patients' airway pressures. PIP is the peak positive pressure detected while the patient is receiving a breath from the ventilator. Elevated PIP (>35 cm H_2O) signifies pulmonary pathology or patient-ventilator malinteraction. High PIPs are caused by either an increase in AR, a decrease in C_p , or extra pulmonary parenchymal pathologies causing increased pleural pressure which is then transmitted to the airways.

When the PIP is elevated the first question is if it is due to increased AR. Some ventilators will calculate and display AR on a breath by breath basis. Values above 4 cm/L/second are elevated. Otherwise, a quick maneuver to assess AR is to have the ventilator hold the V_t in the patient for 1 - 2 seconds. This is usually done through the "inspiratory pause" function of the ventilator. At the end of the pause the ventilator will read out a "plateau pressure" (PP). This pressure should roughly reflect the alveolar pressure, as there is no airflow between the ventilator pressure transducer and the alveoli during an inspiratory pause. If the PP is low, and the PIP is high, the problem is one of AR. If the PP is high, as well as the PIP, the problem is one of poor C_p or increased pleural pressure.

Causes of increased AR include:

- Malposition of the ETT. The ETT can be too high with the tip being in the larynx. The ETT tube can be too low, either up against the carina or in one of the main stem bronchi. Occasionally the ETT can be up against the tracheal wall in patients with airway, abnormalities from, for example, kyphoscoliosis, or a prior extensive lung injury resulting in a torturous trachea.
- Mucus or mucopurulent plugging of the ETT tube or airway.
- Bronchospasm and/or airway edema
- Biting of the ETT

Assessment for these possibilities should include physical

examination with inspection of the ETT tube where it exits the mouth. Look for biting of the tube and tube position. In general, the ETT should be approximately 21 cm at the teeth for an average size woman and 23 cm for an average size man. Auscultation should be performed for rhonchi suggesting airway secretions and wheezing suggesting bronchospasm. Focally decreased breath sounds can signify bronchial obstruction by mucous plugging, as well as pneumothorax and pleural effusion. CXR examination after cardiac surgery is routine and should be checked for the position of the ETT (3 cm. +/- 1 cm from the carina). If, after inspection, auscultation, and review of the CXR the cause of AR is not obvious, a suction catheter should be introduced through the ETT to feel for resistance and to suction possible mucous plugs. If resistance is met, the ETT should be repositioned if poor positioning appears to be the problem. If ETT positioning is not the problem, and the patient has relatively stable gas exchange, then a bronchoscope should be performed through the ETT to inspect the etiology of the airway obstruction. The bronchoscope can sometimes more efficiently clear secretions and guide the position adjustment of the ETT. There are occasions when the airway is obstructed and gas exchange rapidly deteriorates into a dangerous range. In these circumstances it is most appropriate to emergently extubate and reintubate the patient. This should be done by an anesthesiologist or a pulmonologist experienced in difficult airway management.

If PIP elevation is not caused by AR then the problem is pathology causing poor C_p or increased pleural pressure. PP is elevated in both of these circumstances and C_p (static) is decreased. Many modern ventilators will calculate the static C_p , but you can calculate it yourself through the following formula: $C_p = V_t / PP - PEEP$. C_p less than 50 ml./cm H_2O indicates a problem. Lung parenchymal problems causing significantly decreased C_p include any air space filling or interstitial infiltrative processes. In the post cardiac surgery population the most common cause is pulmonary edema from congestive heart failure and/or volume over load. Nonhydrostatic pulmonary edema (adult respiratory distress syndrome) and diffuse, severe pneumonia can markedly decrease lung compliance as well. Increased pleural pressure can be created by chest wall edema, abdominal distention, pneumothorax, pleural effusions, patient ventilator malinteraction, or "intrinsic PEEP". Intrinsic peep (P_i) is a pathophysiology that can occur in patients with obstructive lung disease that can create markedly elevated PPs (32). In this disorder, because of expiratory flow limitation, air is trapped in the lung,

creating large increases in pleural pressure. Evaluation for these possibilities should begin with inspection to look for signs of patient ventilator malinteraction such as patient inspiratory or expiratory efforts out of synchrony with the ventilator cycle. The chest wall and abdomen should be inspected as well. Auscultation for crackles, signifying interstitial or air space filling processes and for decreased focal breath sounds consistent with pneumothorax or pleural fluid should be performed. Again, the postoperative CXR will be an important diagnostic tool to look for these pathologies. To diagnose Pi the ventilator's expiratory port is occluded at the end of expiration, if positive pressure is detected Pi is present. Many recent vintage ventilators have automated programs to do this.

Treatment of specific entities causing elevated PP and depressed Cp will be discussed below. However, while specific underlying pathologies are being treated it is very important to adjust the ventilator to decrease the PIP (ideally to less than 35 cm H₂O) and especially the PP (to less than 30 cm H₂O). Remember the PP reflects the pressure seen by the alveoli. High pressures in the alveoli can cause them to rupture creating a pneumothorax or pneumomediastinum, and are also associated with overdistension of these lung units and consequent injury (sometimes called "volutrauma") (33). The first therapeutic maneuver is to decrease the Vt. If the pressures are still elevated, then, depending upon the ventilator, the peak inspiratory flow rate should be decreased or the inspiratory time increased. Maneuvers thereafter are dependent upon the specific causative problem

VENTILATOR WEAN AND EXTUBATION

Patients are ready to wean when the following conditions are met:

- Anesthesia and neuromuscular blockade have abated and neurologic status is adequate.
- Gas exchange is adequate.
- Hemodynamics are stable.
- There is no evidence of active post-operative bleeding.

At our institution, non-complicated cases are weaned per respiratory therapist driven protocol. To initiate the protocol, the patient must be alert, following commands and able to raise his or her head up off of

the bed without assistance. "Pulmonary mechanics" are also performed to grossly assess neuromuscular function. These are a series of maneuvers done by the respiratory therapist to assess the ventilatory muscle power. At our institution we use two, the maximum inspiratory force (MIF), which is the maximum negative inspiratory pressure that the patient is able to generate, and the vital capacity (VC), which is the maximum amount of volume a patient can expire after taking in as deep a breath as possible. Prior to extubation, we would like to see our patients generate a MIF of greater than or equal to 20 cm. and a VC of >10 ml/kg. Adequate gas exchange is demonstrated by a PaO₂ of >60 on an FiO₂ of <60%. When patients are extubated and removed from positive pressure ventilation, typically, their ventilation-perfusion matching worsens slightly, and Fi H₂O requirement goes up by approximately 10 - 20%. Therefore, generally, we do not extubate patients from higher FiO₂ as it could place them at risk for hypoxemia. PaCO₂ in general should be <45 to extubate. In patients, however, with advanced COPD, the CO₂ is allowed to be near their baseline. Mean arterial pressure (MAP) should be >60 and stable, as organ function below this MAP is compromised, including heart, brain, renal and diaphragm function.

Weaning compromises, in essence, transferring WOB from the ventilator to the patient. There are several ways to do this. At our institution, the SIMV rate is gradually turned down and eventually off, and the patient is placed on only PS. PS of from 8 - 14 cm, is enough to compensate for the WOB through the ventilator circuitry in patients with various types of lung diseases (34). Because of the increased AR and decreased Cp seen routinely in post CS patients (see above section *Alterations of pulmonary physiology following cardiac surgery*), we place our patients on a PS level of 10 cm. The patient is then followed on PS from 30 minutes to two hours after which ABG is checked for hypoxemia or hypercarbia. Just as importantly, though, the rapid shallow breathing index (RSBI) is calculated. This is a parameter developed to predict success of extubation (35). The RSBI is calculated by the equation: Vt (liters)/RR (breaths per minute). If this ratio is <105, then the patient should be successfully extubated.

Prior to actual extubation, patients at our institution are first taught proper coughing technique for when the ETT is removed. This entails use of a specially designed pillow to brace against their sternum when coughing. Then, typically, our respiratory therapists or nurses will suction the patient's mouth and hypopharyngeal areas where

secretions sometimes will pool, as well as suction the airways through the ETT. The ETT is removed while the patient is forcefully expiring. An oxygen mask delivering a FiO₂ of about 10% higher than that used for MV is placed on the patient. The FiO₂ is then gradually dropped to the lowest level that still maintains adequate oxygen saturation over the ensuing minutes to hours. For at least several hours after extubation, we encourage frequent coughing and incentive spirometry use. It is important to be aware that a small percentage of patients will exhibit signs of laryngeal edema after extubation. The edema is caused by ETT trauma to the upper airway. Patients at risk for this development include those whose intubation was difficult, and those who have experienced prolonged MV. Signs of edema may not be immediately obvious, but may be delayed for one to two hours. Clinically, the patient will exhibit signs of respiratory distress with increased respiratory rate, use of accessory breathing muscles. Hoarseness often accompanies this symptomatology and on physical examination, stridor can be appreciated. Treatment is nebulized racemic epinephrine and glucocorticoids (methylprednisolone or decadron) (see *Drug Table 2* at end of chapter). At our institution, we have had some success with the use of a helium oxygen mixture, which allows easier flows through the edematous airway. However, reintubation should be performed if the patient does not exhibit improvement rather quickly, as delay may so compromise the airway, that an ET tube will be unable to be pass through the glottis. If this occurs, an emergent tracheostomy will be required.

VENTILATOR MANAGEMENT FOR PATIENTS WHO ARE NOT QUICKLY WEANED

The majority of cardiac surgery patients are weaned off the ventilator in less than 48 hours. Between 10 and 23% of patients, however, are not. These patients have persistent neurological, pulmonary, cardiac or ventilatory mechanics problems, which require continued MV. The question arises in this circumstance of what is the best ventilator strategy to support these patients until they are ready to be weaned off MV. There is actually very little guidance from clinical studies to answer this question. However, in addition to providing adequate oxygenation and ventilation, two concerns typically inform most recommendations. These are concern for the development of diaphragm and other ventilatory muscle deconditioning, and contrarily, concern that patients may be forced to perform too much

WOB. Animal data suggests that the diaphragm and other ventilatory muscles can certainly become deconditioned within a matter of days (36). Therefore, it is best to allow patients some WOB. Recently, an entity called “iatrogenic ventilatory dependence” has become a concern (37). This occurs when the MV is set in such a way that the patient has a constantly fatiguing load to breathe against. Our institutional approach is usually to use the SIMV mode with PS. RR is set at 8 -12, with a standard V_t (10 - 15 ml/kg). PS is generally set at from 10 - 20 cm H₂O so that the patient initiated breath will be at least 5 ml/kg or 500 cc. The overall respiratory rate, meaning both the SIMV rate and the patient initiated respiratory rate, should be approximately 15 - 25 breaths/minute. This generally will allow the patient an adequate amount of work, but not too much. Often, we will increase the SIMV rate at night to allow the patient rest. So, for example, if we have a patient who has had a post-operative stroke, and whose mental status does not allow rapid weaning from the mechanical ventilator, we will set the SIMV rate at 4 and pressure support at 15 cm during the day, and an SIMV rate of 8 and pressure support of 15 cm at night.

If it appears that the patient will require mechanical ventilation for more than ten days or so, we recommend tracheostomy. This practice is in concordance with recent societal and expert recommendations for tracheostomy in patients who will require prolonged MV (38). The reasons for these recommendations include:

- Glottic protection.
- Patient comfort and the ability to do mouth care.
- A more secure airway.
- Greater ease of weaning from the mechanical ventilator.

There has been some debate in the post CS patients as to where the tracheostomy should be placed. There has been an argument made that the tracheostomy should be a cricothyroidotomy as this incision location is further from the sternotomy wound and may result in less sternal wound infection (39). At our institution, however, we perform tracheostomy in the standard position as we have found this to result in far less difficulties with the airway. Percutaneous tracheostomy is often performed at our institution, but for patients with a thick neck or for other anatomic concerns, a standard tracheotomy is preferred. Most often both procedures can be done at the bedside.

Special Note

Occasionally patients will come out of cardiac surgery with an open chest. This can be either an open sternum with skin closure or a completely open chest with dressing. The reason for this is usually substantial mediastinal bleed at the time of operation, or cardiac edema. These patients require full mechanical ventilatory support and, in fact, also deep sedation, if not neuromuscular blockade, so that the mediastinum is not further traumatized by the sternotomy edges.

Patients who will require prolonged mechanical ventilation (clearly for more than 48-hours) should be started on nutrition. Enteral feeding is preferred and is best be instituted with the placement of a soft, distally weighted feeding tube, placed through the nose. These tubes are designed to be floated through the stomach and to have their distal tip in the duodenum. Such tubes decrease the patient's risk of lung aspiration of feeds. High nutritional tube feeding formulas are typically started at a low rate of 10-20 ml/hour which is advanced over the next 24 hours to a target rate which provides the estimated caloric and protein needs of the patient. We often begin patients on mecloramide concurrently with the initiation of enteral tube feeds if the patient has hypoactive bowel sounds. If patients cannot tolerate tube feeding, then total parenteral nutrition (TPN) is begun through a central line. Patients requiring prolonged mechanical ventilation are also prone for stress gastritis in as much as 40% of the patients (40). Prophylaxis can be accomplished with fairly equal efficacy by H2 blockers such as famotidine, proton pump inhibitors such as lansoprazole, and sucralfate. Among these, sucralfate appears to be associated with a lessened incidence of nosocomial pneumonia by preserving the gastric acid barrier between enteric pathogens and the lung (41). Incidence of nosocomial pneumonia is substantial at 3% per day of mechanical ventilation (42). Therefore, it is prudent to be vigilant to its development by noting the quantity and quality of secretions and monitoring chest x-rays. Patients on mechanical ventilation are also at substantial risk for the development of deep venous thrombosis (DVT) in the legs. This is somewhat less of a concern in post CS patients for the immediate postoperative period, as reports of DVT and PE are rare in this time frame. This is presumed to be because of the anti-thrombogenic effect of cardiopulmonary bypass. However, incidence of thromboembolic events appears to approach that of other patient populations on MV beginning around fourth postoperative day (18). Our patients typically have support hose in the immediate post-operative period

and, as they get out from CPB, we will typically use a low molecular weight heparin or pneumatic compression devices as prophylaxis.

SPECIFIC PULMONARY PROBLEMS FOLLOWING CARDIAC SURGERY

HYPOXEMIA

Hypoxemia is defined as a pressure of oxygen in blood (paO_2) of less than 60 mm Hg, or a hemoglobin O_2 saturation of less than 90%. The differential diagnosis of pathologies causing post CS hypoxemia is:

- Alveolar hypoventilation.
- Ventilation / Perfusion (V/Q) mismatch caused by the following clinical pathologies:
- Airway obstruction caused by ETT compromise, bronchospasm, or airway secretions
- Hydrostatic pulmonary edema
- Adult respiratory distress syndrome (ARDS)
- Pneumonia
- Atelectasis
- Pulmonary embolism
- Hemodynamically active drugs
- Post operative pulmonary hypertension
- Decreased mixed venous oxygen (MVO_2)
- Anatomic right heart to left heart shunting of venous blood.

If the lung's alveolar ventilation (V_a), as a whole, is too low, more O_2 is removed from alveolar air by capillary blood than is replenished, resulting in a low alveolar pressure of O_2 (pAO_2). Since pAO_2 is the driving pressure of O_2 into capillary blood, this in turn results in a low paO_2 . Similarly, when V_a is low, CO_2 is not removed from the alveoli and paCO_2 rises above the normal value of 40 mm Hg. So, the first step in evaluating a patient who is suspected of being hypoxemic or known to be so from O_2 saturation data is to obtain an ABG. If indeed the paO_2 is low and the paCO_2 is high, then the hypoxemia is very likely to be, at least in part, due to alveolar hypoventilation. Marked increase of pulmonary "dead space" can also increase paCO_2 , but this circumstance is very unusual, and discussed below in the section on *Hypercarbic Rrespiratory Failure*. Whether or not the hypoxemia is solely due to alveolar hypoventilation can be determined by utilizing the relationship between pAO_2 and paCO_2 in

the “alveolar gas equation”, which states:

$$pAO_2 = PIO_2 - paCO_2/R - F,$$

where PIO_2 is the pressure of O_2 in inspired air, R is the respiratory quotient (number of moles of CO_2 produced per mole of O_2 consumed), and F is a small correction factor. PIO_2 can be derived from the FiO_2 , barometric pressure (pB), and water vapor pressure (pH_2O). At sea level the FiO_2 of room air is 0.21, pB is 760 mm Hg and pH_2O is 47 mm Hg (at 37°C). Therefore $PIO_2 = 760 - 47 \times FiO_2 = 713 \times 0.21 = 150$ mm Hg. R is almost always 0.8 under most physiologic conditions and F is so small that it can be ignored. So, on room air, at sea level, $pAO_2 = 150 - paCO_2/0.8$. Normally the pAO_2 should be within 7 mm Hg + 0.27 x patient’s age of the PaO_2 . If the pAO_2 - the paO_2 (alveolar-arterial oxygen difference (A-a O_2D)) is greater than this, then there is one of the other major pathophysiologic causes of hypoxia at play as well, specifically V/Q mismatch, anatomic right heart to left heart shunting of venous blood, or impairment of O_2 diffusion across the alveolar capillary membrane.

For example, if a patient’s O_2 saturation is 85% on room air, the first step in the evaluation is to obtain an ABG. If the ABG shows a paO_2 of 50 mm Hg, and $paCO_2$ of 80 mm Hg, you know immediately that this patient’s hypoxemia is very likely to be, at least in part, due to alveolar hypoventilation. To determine if there are other pathophysiologies at play, the alveolar gas equation is used to calculate this patient’s pAO_2 . $150 - 80/0.8 = 150 - 100 = 50$. A-a O_2D is therefore 0, and alveolar hypoventilation is the sole cause of the hypoxemia.

The A-a O_2D can be calculated when patients are on different FiO_2 s, but there are two caveats to doing so. First, when external O_2 is applied to a patient it is difficult to know the exact FiO_2 the patient is breathing because the patient will almost certainly entrain some room air with inspiration. Secondly, as the FiO_2 increases so too does the anticipated A-a O_2D (43). Specific clinical entities causing alveolar hypoventilation are discussed in the section below on hypercarbic respiratory failure. Under normal physiologic conditions, a patient’s pulmonary capillary flow (perfusion), is matched, or goes to, alveoli with good ventilation. When a significant amount of perfusion goes to relatively poorly ventilated alveoli (V/Q mismatch) the result is hypoxemia. Most post cardiac surgery hypoxemia is caused by this

pathophysiology.

Airway obstruction results in poorly ventilated lung units and hypoxemia. Obstruction in post CS patients is commonly caused by ETT malposition (discussed above under *Ventilator adjustment and trouble shooting*), “plugging” of airways by mucous or mucopurulent secretions, and bronchospasm. Patients with the chronic bronchitis variant of COPD are at particular risk for airway plugging. These patients, as a baseline, produce large quantities of mucous, and often, because of impaired pulmonary mechanics, have a poor cough. Anesthesia, analgesia, and the pain of coughing associated with sternotomy often precipitate the build up of obstructing mucous in chronic bronchitis post cardiac surgery. Post operative patients who develop acute bronchitis with its attendant large quantities of mucopurulent secretions, for the same reasons are at risk for airway compromise. Bronchospasm usually occurs in patients with underlying obstructive lung disease, either asthma or COPD, and may be triggered by a variety of perioperative factors. Among those factors are bronchial edema associated with the volume overload attendant to CPB, irritation of the airway by the ETT and suctioning, aspiration of gastric contents at the time of anesthesia, acute bronchitis, and very rarely an adverse medication reaction from beta blockers or narcotics, which can cause histamine release. Chest auscultation findings of patients with significant mucous or mucopurulent secretions include rhonchi and expiratory crackles, as well as focally decreased breath sounds in areas where bronchi have been completely occluded by secretions. Patients with bronchospasm demonstrate expiratory and occasionally inspiratory wheezes. Therapy for airway secretions includes mechanical suctioning through an ETT if the patient is intubated or nasotracheal suctioning if the patient is not. Mucolytics are helpful, delivered either through nebulized acetylcysteine (which, it is important to realize, can prompt bronchospasm), or enterally using guaifenesin, most commonly (see *Drug Table 3* at the end of the chapter). We avoid vigorous chest percussive therapy, at least in the immediate postoperative period, for fear of disrupting the sternotomy. Patients are encouraged to cough, however, while bracing their sternotomy with a specially designed pillow. Bronchodilators help, as well, in promoting cough and allowing secretions to be mobilized. Of course, if acute bronchitis is at play we treat with antibiotics. For bronchospasm, the cornerstone of treatment is inhaled bronchodilators, which basically are of the beta 2 - adrenergic agonist or anticholinergic drugs. The most common beta - agonist is albuterol in either standard form or more

recently developed (R) isomer form (levalbuterol). Levalbuterol has been shown to have a longer half-life, to be a more effective bronchodilator and have less cardiac effects (44). The only anticholinergic currently available is ipatropium bromide. We typically use both albuterol and ipatropium bromide to maximize bronchodilation. We avoid intravenous catecholamines and theophylline in managing bronchospasm because of the cardiac side effects. *Drug table 1* (at end of chapter) lists various regimens for bronchodilating drugs. Bronchitis with the bronchospasm are treated with antibiotics. Patients with hydrostatic pulmonary or bronchial edema may also benefit from diuretics.

Many patients post-cardiac surgery, have an element of hydrostatic pulmonary edema (Conti 45). Hydrostatic pulmonary edema in the post-cardiac surgery patient is caused by a combination of the large volumes of fluid given to the patient to maintain CPB, the drop in oncotic pressure of the blood caused by the hemodilution of blood necessary for cardiopulmonary bypass, and sometimes cardiac dysfunction caused by either temporary stunning of the myocardium in CPB or congestive heart failure (46). Hydrostatic pulmonary edema is characterized by physical exam findings of end-inspiratory lung crackles especially at the lung bases, CXR demonstration of pulmonary venous congestion and a prominent interstitial pattern, A-a O₂D elevation and decreased pulmonary compliance. Treatment of hydrostatic pulmonary edema is diuresis. This is the most effectively accomplished by standard loop diuretics.

Fully developed ARDS is a relatively rare occurrence following CPB but is associated with a mortality of 40 - 60% (15). ARDS can be caused by systemic inflammation prompted by CPB machinery membranes, aspiration of gastric contents at the time of anesthesia induction and, in the instance of multiple blood product transfusions perioperatively, transfusion related acute lung injury (TRALI).

The exact mechanism for the development of systemic inflammation associated with CPB is not completely clear. However, it appears to involve complement activation, which prompts neutrophil recruitment to the lung. These activated neutrophils, through release of toxic granular contents, are thought to be the cause of "pump lung" (15). It does appear that, though frank ARDS occurs in only 1 - 2% of CPB cases, a lesser version of lung injury associated with this inflammation is far more common, occurring in 12% of patient in a recent series (47).

Aspiration of gastric contents is also rare, but still does occur and can cause ARDS. Usually the anesthesiologist will have noted this occurrence at the time of induction, though not always. TRALI is generally caused by antibodies in the donor blood to the patient's native white blood cells, resulting in clumping of white cells in the lung, the so-called leucoagglutinin reaction. These clumped white cells are activated and produce inflammation of the lung and ARDS. Steroids appear to be helpful for this pathology in our and written experience (48). Very rare is a reaction to protamine which is given at the end of CPB to reverse heparin, involving an anaphylactoid mechanism, which can involve the lung in ARDS (49).

ARDS is clinically characterized by end-inspiratory crackles on lung auscultation, chest x-ray evidence of a diffuse interstitial and alveolar filling pattern, decreased lung compliance, and an elevated A-a O₂D. Specific treatment of the acute phase ARDS is essentially supportive care. This means maintenance of mechanical ventilation until hopefully the ARDS resolves. It is very important, however, to properly set the mechanical ventilator. Recent studies have clearly shown the advantage of ventilator strategies which avoid of "overdistention" and "underdistention" of the lung (50). This is because in this, and many other types of lung injuries, there are lung units that are minimally affected and lung units which are severely involved. Vt will typically go to the less affected and more compliant lung units. Traditional sized Vt of 10-15 ml/kg can overdistend these lung units and injure them creating a spiral of worsening ARDS. Recent studies using a Vt of between 5 and 10 ml/kg, and not allowing PPs to exceed 30 cm of H₂O pressure have shown significant improvements in mortality and other outcomes of patients with ARDS (51, 52)

Part of the pathology of ARDS is injury to the type II alveolar cells, which produce surfactant. Without surfactant, many areas of the lung collapse. These "underdistended" lung units, are continually being opened by traditional volume or pressure cycled mechanical breaths only to collapse again upon expiration. This continual opening and closing creates destructive sheer forces to the alveolar membrane, and escalates lung injury, just as alveolar overdistension does. PEEP,

originally developed to recruit collapsed lung units for ventilation to improve oxygenation, appears to be the most effective tool for prevention of this form of ventilator toxicity. The dose of PEEP is best judged utilizing volume pressure curves and inflection points (51). Discussion of these techniques is beyond the scope of this text. However, for practical purposes at the bedside, titrating PEEP in the traditional fashion for O₂ improvement will result in a dose that has good effect in preventing “underdistension”. We recommend starting with a PEEP of 3 cm. and go up in increments of 2 cm, not exceeding a PEEP of 15 cm. Follow arterial oxygenation to see that it is improving with increments in PEEP, as well as lung compliance. It is important to carefully follow the patient’s blood pressure, as eventually, by increasing PEEP, cardiac output will become compromised. The dose of PEEP where oxygenation and compliance is best and where cardiac output is not compromised, is selected. This is usually between 5 and 12 cm of PEEP.

ARDS is now known to have a biphasic natural history. After the acute “inflammatory” phase the lung will enter either a resolution and reparative phase, or what is called now the “fibroproliferative” phase. This phase is marked by the influx of fibrosis producing inflammatory cells and destruction of the lung’s capillary bed. Clinically, the fibroproliferative phase of ARDS is characterized by low-grade temperatures, progressive and severe decline in pulmonary compliance, a progressive increase in dead space, and gradually worsening CXR. This is accompanied by a need for increased minute ventilation to maintain adequate gas exchange. This phase of ARDS usually occurs three to seven days into the course of the disease, and there is now good evidence that steroids are a very effective therapeutic tool for fibroproliferative phase ARDS (53), and in our hands, has proved to be quite helpful. It is very important to make sure that there is no active infection, however, before instituting steroids.

Pneumonia and atelectasis provoke hypoxemia by causing affected

lung units to be poorly ventilated. Pneumonia is discussed below (*Bronchitis and Pneumonia*). Atelectasis in CS patients is expected, to some degree, as part of the normal postoperative course (see *Alterations of Pulmonary Physiology Following Cardiac Surgery*). Additional problems and pathologies contributing to, or causing atelectasis include phrenic nerve dysfunction, pleural effusions, and splinting from postoperative pain. Physical examination is characterized by again end-inspiratory crackles, though these crackles typically are higher pitched, or “dry”. CXR demonstrates low lung volumes and sometimes, obvious effusions, segmental or lobar lung collapse, or “plate-like atelectasis”. Treatment of postoperative atelectasis consists, of encouraging cough, incentive spirometry, mobilization, and adequate pain control. We avoid the use of positive airway pressure devices such as IPPB, or IPV out of concern for disrupting the sternotomy. For patients on the ventilator, maneuvers to recruit alveoli consist of increasing the tidal volume, while of course watching the PIP, and utilizing PEEP in the same fashion as would be utilized for ARDS but at generally much lower levels.

Pulmonary embolism used to be thought of as a rare occurrence in the CS patient. However, the incidence in the largest series to date was 3.2% (18). Interestingly, all events occurred after the first three postoperative days. This is presumably because of the coagulopathy created by CPB. Important risk factors for the development of PE were prolonged recovery from surgery, obesity, and heparin induced thrombocytopenia. To prophylaxis against thromboembolic events it is our practice to use graded compression stockings for all CS patients in the immediate postoperative period, but out of concern for bleeding we do not use heparin until after the third postoperative day. Suspicion for pulmonary embolism, should occur when there is an abrupt worsening of oxygenation without an obvious cause. Physical examination findings include tachypnea, a split P2 upon cardiac auscultation, and jugular venous distension. CXR will often be clear, or unchanged, but occasionally demonstrate oligemia in one area of the lung, or a peripheral wedge shaped opacification signifying an

infarct. A-a O₂D will be elevated. Echocardiogram can be very helpful in assessing for the elevated pulmonary artery pressures and right ventricular strain that accompany large or multiple emboli. Diagnosis of PE usually requires a ventilation-perfusion scan, CAT scan of the lung utilizing special imaging techniques, or pulmonary arteriogram. Therapy of pulmonary embolism is anticoagulation, usually with heparin. Rarely, if a pulmonary embolism is massive and proximal in the pulmonary artery, the patient will require mechanical disruption or removal of the embolism by catheter techniques or surgical thrombectomy. In the immediate postoperative period lytic agents, such as TPA, are contraindicated. Hemodynamically active drugs have effects on the pulmonary vascular bed as well the systemic circulation. By thus altering perfusion patterns in the lung, V/Q mismatch can be created with resultant hypoxemia. Therefore, it is wise to monitor for adverse oxygenation effect while using vasoactive drugs, especially vasodilators. Postoperative pulmonary hypertension (fully discussed below in *Postoperative Pulmonary Hypertension*) causes hypoxemia by causing V/Q mismatch, predominantly, but also occasionally by prompting opening of the foramen ovale.

Decreased mixed venous oxygen occurs in patients who have decreased O₂ delivery or markedly increased peripheral oxygen utilization. For practical purposes, this happens in post cardiac surgery patients who have impaired cardiac output and/or a high metabolic rate (usually from a major infection). Under these circumstances peripheral tissue oxygen extraction markedly increases resulting in a low mixed venous (pulmonary arterial) O₂. This blood does not have adequate time to be fully oxygenated by the alveolar capillary unit, and so results in pulmonary venous blood that hypoxemic. Diagnosis of this pathophysiology as a cause of or contributor to hypoxemia is made presumptively under the right clinical circumstances and through mixed venous sampling. This is done most effectively through a pulmonary artery catheter. Therapy for this circumstance involves either increasing O₂ delivery or decreasing peripheral O₂ consumption. Increasing O₂ delivery usually involves improving cardiac output through pharmacological or mechanical augmentation, but also improving O₂ carrying capacity by

increasing intravascular volume and hemoglobin, when necessary. Decreasing O₂ consumption usually involves gaining control of infections and decreasing body temperature through acetaminophen and mechanical cooling devices.

Shunting of venous blood from the right side of the heart to the left heart without passing through the capillary bed for oxygenation requires both an anatomic conduit from right to left, and elevated pulmonary artery pressures. Surgery for CHD would, of course, be the most common setting for this to occur. Otherwise, as mentioned above, if pulmonary artery pressure rises severely or abruptly, an anatomically closed foramen ovale can become patent. Diagnosis of right to left shunting is best made with a contrast enhanced Echo Doppler study. Therapy for shunt begins with maneuvers designed to reduce pulmonary hypertension (see *Postoperative Pulmonary Hypertension*). We have found nitric oxide to be particularly helpful in this regard. Additional temporizing medical maneuvers include using systemic vasopressors to increase left sided cardiac pressures to discourage right to left shunting. Further treatment depends upon the anatomy of the shunt and discussion of such is beyond to scope of this article.

HYPERCARBIC RESPIRATORY FAILURE

Hypercarbic respiratory failure characterized by a pCO₂ >45 mm Hg is almost always caused, in the post CS patient, by alveolar hypoventilation. The differential diagnosis of alveolar hypoventilation in the post CS patient is:

- Decreased central respiratory drive.
- Phrenic nerve injury.
- Mechanical disruption of the chest wall.
- Peripheral neuromuscular dysfunction.
- Airway obstruction.

Decreased central drive to breathe in the post-cardiac surgical patient is nearly always due to over sedation. Rarely a perioperative cerebrovascular accident (CVA) involves the respiratory control center in the medulla of the brainstem. Occult or untreated hypothyroidism can cause an inadequate central drive in response to respiratory load

(54), and is worth checking for in cases of unexplained hypercarbia. A small percentage of patients with sleep apnea will chronically retain CO₂. These patients are usually obese, and have some obstructive lung disease as well (“obesity hypoventilation syndrome”).

Phrenic nerve injury, as mentioned above, is fairly common post CS especially when those surgeries involve CPB with direct cold cardioplegia. A recent, very sophisticated study involving electrophysiologic testing of post-cardiac surgical patients for phrenic nerve dysfunction found it to be present in 26% of patients (55). Eight-five percent of the time, the phrenic nerve abnormality was on the left. All of the remainder of the patients except one had right phrenic nerve abnormalities. One patient had bilateral phrenic nerve abnormalities. In this study, patients were followed for up to one year post-cardiac surgery. In all patients, the phrenic nerve abnormalities completely resolved, usually within the first several weeks of the post-operative period. Unilateral phrenic nerve palsy should by itself not be enough to cause hypercapnic respiratory failure as we know from other studies that it impairs vital capacity by only approximately 25% (56). However, if unilateral phrenic nerve palsy and consequent unilateral diaphragm dysfunction is coupled with other pathologies, such as moderate-to-severe chronic obstructive pulmonary disease, it can certainly produce hypercapnic respiratory failure. Bilateral phrenic nerve palsy would be expected to produce hypercapnic respiratory failure. Though this is rare, it should be looked for in the post-cardiac surgical patient with otherwise unexplained hypercapnia. Phrenic nerve dysfunction is most easily evaluated by the “sniff test”. This test involves having the patient sniff while imaging the diaphragms by fluoroscopy or ultrasound. Lack of diaphragm motion or paradoxical diaphragm motion, meaning the diaphragm moves cephalad rather than caudally, suggests phrenic nerve dysfunction. There is no specific treatment for phrenic nerve dysfunction other than good supportive care and otherwise unloading the ventilatory system from other burdens such as airway obstruction or secretions.

Mechanical chest wall disruption in the post-cardiac surgical patient for practical purposes means a sternotomy that has become unstable causing the patient to exhibit flail chest physiology. Clinically, this is characterized by retraction of the sternum during inspiration. Often, a flailed chest is accompanied by pulmonary secretion build-up as the patient’s cough is very compromised. Therapy for flail chest, of course, involves surgical correction.

Peripheral neuromuscular dysfunction can rarely complicate the post-cardiac surgical course by impairing ventilator muscle function. This can occur when there are severe abnormalities of certain electrolytes, which are necessary for normal muscular function, to include potassium, magnesium, phosphorus and calcium. As mentioned above, rarely post-cardiac surgery, patients will develop systemic inflammatory response syndrome (SIRS). This syndrome can involve peripheral nerves and muscles in what has been called “neuropathy and myopathy of critical illness” (57). There is no specific treatment for these pathologies and they are very difficult to diagnose in the post-cardiac surgical patient. A common cause of hypercapnic respiratory failure in post-cardiac surgical patients is airway obstruction. For patients with advanced chronic obstructive pulmonary disease, the increased airway resistance attendant to CPB can be enough of an additional respiratory load to prompt respiratory failure. Patients with chronic bronchitis often have difficulty clearing their secretions secondary to impaired cough due to the pain of sternotomy. On physical examination, these patients will often be seen using accessory respiratory muscles and demonstrate decreased breath sounds suggesting airflow obstruction. Wheezes and rhonchi will often be auscultated suggesting bronchospasm and airway secretions. Therapy for these patients involves inhaled bronchodilators, the beta-adrenergic agonist and anticholinergic classes as well as mucolytics (see *Drug Table 1* and *3* at the end of chapter). Endotracheal or nasotracheal suctioning is often required to clear airway secretions. We avoid vigorous chest physiotherapy and devices like IPPB and IPV, out of concern for disrupting the sternotomy.

Indications to place extubated patients back on the ventilator include hypercarbia with a pH of less than 7.3 and impending respiratory failure secondary to reparatory muscle fatigue. Some helpful clinical signs of respiratory muscle fatigue are: rapid shallow breathing (RR greater than 35 with small Vts), use of accessory respiratory muscles, and “abdominal paradox” (abdomen goes in rather than distends with inspiration). Sometimes reintubation can be avoided through the use of noninvasive positive pressure ventilation, and it is often worth a trial. We have had good experience with BiPAP applied through a nasal mask. Patients must, however, be alert and able to coordinate with the machine.

As mentioned above, hypercarbia is almost always caused by alveolar hypoventilation. Occasionally, however, a tremendous increase in physiologic “dead space” can cause hypercarbia. Dead space occurs when areas of the lung have more ventilation than they do perfusion. When dead space becomes very, very large, there is not enough capillary bed in contact with the ventilated lung to adequately release blood CO₂. This occurs clinically in the circumstance of massive pulmonary emboli, advanced adult respiratory distress, and severe pulmonary hypertension. Diagnosis and therapy of these entities are discussed in the above section dealing with hypoxemia.

PLEURAL EFFUSIONS

Pleural effusions are common after CS. This is especially so after CABG, where the reported incidence is 40 to 90% in the immediate post operative period, depending upon the series (12,13). In most, though not all reports, bypass grafting utilizing internal mammary arteries (IMAs), rather than just saphenous venous grafts, has a significantly higher incidence of effusions (58). Heart failure or volume overload can certainly cause pleural effusions, but most instances of effusion in the immediate post operative period are not caused by such (13), and their exact etiology is unclear. Proposed mechanisms for the effusions include: a variant of the post cardiac injury syndrome where a local, pleural immunologic reaction produces the effusion, disruption of lymphatic drainage of the pleural space, especially when IMAs are harvested, leakage of mediastinal fluid through pleurotomy (often created in the process of IMA grafting) (14), or a reaction to topical hypothermia (59).

Most effusions are small, left sided, and of no clinical consequence (14). However, up to 10% of effusions are moderate to large and require treatment, for symptom relief (60). Early postoperative effusions are bloody exudates and eventually resolve after one or two thoracenteses, though they may persist in minimal quantity for several months. Rarely, effusions continue to recur after several thoracenteses. These effusions are exudative and typically contain lymphocytes as the predominant cell type (61). These patients can be tried on nonsteroidal anti-inflammatory agents and/or glucocorticoids (typically prednisone). In our experience, pleurodesis, best achieved through thoracoscopy is often required to permanently control the effusion.

Two other mechanisms for the development of post CS pleural effusions are worth noting. The post cardiac injury syndrome can occur after myocardial infarction, cardiac trauma or surgery (62). It is characterized by fever, positional and pleuritic chest pain, pericardial and pleural effusions, often pulmonary and parenchymal infiltrates, and leukocytosis. The syndrome occurs in 10 - 40% of cardiac surgery patients, depending upon the series. Time to onset is one to several weeks after surgery with three weeks as an average. Antimyocardial antibody can be helpful in diagnosis though not definitive. Treatment is

NSAIDs and/or glucocorticoids. There have been rare reports of chylothorax after cardiac surgery (63). These presumably result from trauma to the thoracic duct. Concern for chylothorax should be raised when pleural fluid is milky in appearance. Diagnosis is established by high levels of triglycerides in the pleural fluid. In most instances, with TPN and time, the chylothorax will resolve. Rarely, corrective surgery or pleuroperitoneal shunting will be required.

BRONCHITIS & PNEUMONIA

The incidence of postoperative pneumonia varies between 3 and 22% (64,65). The most recent series, using sensitive and specific diagnostic tools such as the protected microbe collection brush, place the incidence at between 5 and 10% (17). Occurrence of this nosocomial pneumonia peaks on postoperative day four (66), though we and others have seen well-developed cases on the first postoperative day. Interestingly, there is essentially no literature specifically addressing bronchitis in post cardiac surgery patients. This, presumably, is because of the difficulty of making a certain diagnosis of bronchitis in these patients. However, in our experience it is also a prevalent problem, and if not addressed leads to pneumonia. Pneumonia in post cardiac surgical patients is associated with a higher mortality and prolonged ICU and hospital stays (17).

Several factors place certain cardiac surgery patients at a greater risk for the development of pulmonary infections. These include a history of COPD, reintubation after initial weaning and extubation from the ventilator, mechanical ventilation of more than two days duration, recumbent position in the first 24 hours post-op, nasogastric tube, the use of H2 blockers and antacids, use of perioperative broad spectrum antibiotics, multiorgan dysfunction and/or high APACHE III score, and multiple perioperative transfusions of blood products (64,67,68,69,66,17). These risk factors are not surprising in light of our understanding of the pathophysiology of nosocomial pneumonia. The key initial step in hospital-acquired pneumonia is colonization of the upper airway, i.e. oropharynx and larynx, with pathogenic bacteria (70). These pathogens then gain access to the lower airway through microaspiration, especially in patients with poor cough. Impaired local and systemic defense systems give virulent organisms

the advantage to establish infection in the bronchi and lungs. Patients with COPD, of course, and those who require reintubation because of inadequate pulmonary mechanics, have an impaired cough. This, together with postoperative pain and analgesia, allows the development of bronchitis and/or pneumonia. We know from studies in non CS patients that prolonged MV is associated with a high risk for pneumonia. One recent study placed the incidence at about 3% per day. Endotracheal tubes allow secretions from the upper airway and from the GI tract, containing large numbers of microbes, to pool in the upper airway. These secretions will occasionally slip past the Eli cuff into a lower airway defense system that is compromised by the trauma of the ETT and suctioning, Recumbent position, nasogastric tubes, and medications which decrease the acidity of the stomach allow reflux of GI tract microbes up the esophagus into the airway. Broad-spectrum antibiotics is associated with the development of late nosocomial pneumonia with, as would be expected, resistant organisms (71). Patients with multiorgan dysfunction and high APACHE scores are known to have impaired immune function (68), and blood transfusion has also been shown to lower immunity (72).

Prophylaxis against pulmonary infections

These data, and our experience, have led to practices at our institution aimed at decreasing the incidence of postoperative pulmonary infections. We pay special attention to, and augment pulmonary toilet in patients with COPD through the use of bronchodilators, mucolytics, incentive spirometry, cough encouragement (or frequent suctioning if on mechanical ventilation), and adequate pain control while avoiding over sedation. Whenever practical, we keep the head of our patient's bed up at least 15 degrees and avoid NG tubes. We use sucralfate, again when practical, when stress gastritis prophylaxis is indicated. Additionally, it is helpful to understand that CS patients routinely experience dysfunctional swallowing for approximately eight hours post extubation (73). A prolonged, severe dysfunction is seen in 4% of the patients. It is usually associated with a very high incidence of pulmonary aspiration and pneumonia (74). Therefore, our protocols keep patients NPO for eight hours after extubation, and when oral intake is begun our staff is trained to monitor for aspiration. Several novel approaches to prevention of pneumonia in the post cardiac surgery patient population are being explored. Some institutions have begun using an ETT which comes equipped with a special catheter for continual suctioning of secretions above the ETT

cuff (75). At our institution we have shown that chlorhexadine gluconate oral rinses significantly decrease the incidence of oropharyngeal colonization with nosocomial pathogens (manuscript in submission).

The classic clinical presentation of pneumonia usually includes fever, localized inspiratory crackles upon auscultation, focal infiltrate on CXR, leukocytosis, and production of purulent appearing sputum, the gram stain of which demonstrates many neutrophils and few epithelial cells, and a dominant organism. Unfortunately, there are other factors confounding the diagnosis of pneumonia in the postoperative patient. Pulmonary edema or ARDS can make physical examination and CXR findings difficult to interpret. Fever and leukocytosis are common in the immediate post operative periods as well. In these circumstances we have found sputum gram stain and quantitative sputum cultures to be invaluable.

Presentation of bronchitis also includes fever, purulent sputum, expiratory crackles, and rhonchi. Nosocomial pneumonia, in general, carries with it a mortality of 30 to 50%, but multiple studies have shown that empiric antibiotic therapy significantly lessens these numbers (71). Therefore, when pneumonia or bronchitis is clearly present, or strongly suspected, we culture the sputum and begin empiric antibiotics. Recent series have shown that the predominant organisms responsible for pneumonia in cardiac surgical patients are gram-negative enteric bacteria (17). *Staphylococcus Aureus*, however, has become a major pathogen in recent years accounting for nearly 19% of infections in a recent study (17). Empiric antimicrobial therapy, therefore, usually consists of a third or fourth generation cephalosporin and perhaps vancomycin. It is highly advised, though, to know the spectrum and common pulmonary nosocomial pathogens and their sensitivities at your institution to guide your empiric antibiotic choice. When sputum culture and sensitivities data become available it is very important to narrow the spectrum of antimicrobials as much as possible. Treatment involves, as well, application of good pulmonary toilet involving frequent coughing or suctioning, mucolytics, and bronchodilation if bronchospasm is present.

POSTOPERATIVE PULMONARY HYPERTENSION

Pulmonary hypertension is common after CS, as is depression of right ventricular function (76). These effects, for most patients, are mild,

last only hours, and do not significantly effect clinical course or outcome (77). However, patients with severe biventricular failure, and in those undergoing repair of congenital heart disease, mitral valve replacement, orthotopic heart transplant, or implantation of left ventricular assist devices are at risk for the development of severe pulmonary hypertension, and consequent right ventricular decompensation and death (78).

Pulmonary hypertension post cardiac surgery appears to be mediated by pulmonary vascular endothelial injury and dysfunction, which prompts vasoconstriction of the pulmonary vascular bed (20). A variety of factors can promote such injury and dysfunction, prominent among which are: intra and post operative hypoxia, hypercarbia, acidosis, intrinsically produced or extrinsically administered catecholamines, blood vessel wall shear stress associated with increased blood flow in conditions of left to right intracardiac shunt, and pathophysiologic changes sometimes attendant to CPB including pulmonary leukosequestration, production of inflammatory mediators, and, perhaps most powerfully, ischemia and reperfusion injury of the pulmonary vascular bed. Patients who go into CS with an already elevated pulmonary vascular tone, from, for example, advanced mitral valve disease, severe left ventricular dysfunction, or congenital heart disease, are particularly susceptible to these effects. When pulmonary vascular resistance markedly increases in these patients it can overburden an often already dysfunctional right ventricular leading to RV distension. This increases RV free wall tension and oxygen consumption as well as decreasing coronary artery perfusion. This sequence can spiral the RV into complete decompensation resulting in poor LV filling and cardiogenic shock.

Protamine, administered to reverse heparin effect at the end of CPB, can rarely cause a tremendous, abrupt rise in pulmonary vascular tone, which can produce right ventricular failure (79). The exact mechanism of this idiosyncratic reaction is unclear but has been suggested to involve complement activation and /or cyclooxygenase products.

In patients at risk for the development of deleterious pulmonary hypertension, avoiding hypoxia, hypercarbia, high mean airway pressures and acidosis is very important in preventing or at least lessening, this complication. A number of other experimental strategies for preventing pulmonary hypertension are being explored in these patients to include use of ultrafiltration during CPB,

“substrate enhancement” with fructose-1, 6 diphosphate (FDP), and the addition of antioxidants during CPB (20).

To effectively treat pulmonary hypertension, and prevent RV failure, it is critical to recognize its development early. This, of course, requires vigilance for its appearance in patients at risk. Physical findings of significant pulmonary hypertension include hypotension, jugular venous distension, a loud split P2, a parasternal or subxyphoid heave, an S3 or S4 heart sound which may vary in intensity with inspiration, hepatic distension and peripheral edema. The CXR will often show clear lung fields and an enlarged cardiac silhouette. Hypoxemia is caused by V/Q mismatching. A pulmonary artery catheter can be an invaluable diagnostic tool in this setting. It will show elevated pulmonary artery pressure as well as elevated right ventricular diastolic and right atrial pressures. In the setting of primary RV dysfunction without pulmonary hypertension, the pulmonary artery pressures will not be elevated. Echocardiography with Doppler can additionally be extremely helpful, as well, by demonstration of elevated PAP and RV pressure and volume overload, often concurrently with an underloaded LV.

Treatment involves therapies aimed at both reducing pulmonary vascular resistance (PVR) and supporting right ventricular function. Initial maneuvers to decrease the PVR include hyperventilation to a paCO_2 of 30 mm Hg, (80), and correction of hypothermia. Thereafter, vasodilators, such as NTG, sodium nitroprusside, calcium channel blockers, tolazoline, PGE2, and PGI2 can be administered through a central venous line, ideally with pulmonary artery catheter guidance, to decrease PAP. Of these, PGI2 appears to be the most effective (81). However, use of these vasodilators is often limited by accompanying systemic hypotension. Inhaled nitric oxide (NO) has proven to be an extremely effective tool for reducing PAP, and does not significantly affect systemic blood pressure. Indeed, recent studies have shown it to be even more effective than PGI2 (82). Currently NO is available to adults only through investigational protocol, but is soon expected to be released for use in CS patients.

Initial investigations into the use of inhaled PGI2 for CS patients has shown great promise as well, but requires more extensive investigation and development. Support of RV function importantly involves maintaining adequate RCA perfusion by maintaining good aortic pressure through appropriate LV preload, and when necessary, pharmacologic inotropic and systemic blood pressure support as well as intra-aortic balloon pump counterpulsation (83). TEE can be used to assess adequacy of coronary artery blood flow (84). RV function can additionally be augmented by appropriate preloading, while avoiding over distension, and use of pharmacologic inotropic support. If these measures fail, the last line of support for the RV is a right ventricular assist device (RVAD).

DRUG TABLES

See Attachment

Table 1

Table 2

Table 3

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