MEDICAL EDUCATION SYSTEMS, Inc.

Course 102

ICU CRISIS MANAGEMENT

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SECTION I - CRITICAL CARE ASSESSMENT

LEARNING OBJECTIVES

Upon completion of this section, given an open-book, multiple choice exam, you will be able to apply the information learned to correctly answer a minimum of 80% of the test items. Successful completion of this exam will require you to have mastered the following learning objectives:

1. Discuss the goals of the critical care areas.

2. Describe the indications and the positive physiological effects for each of the following modalities.

   a. SIMV
   b. PEEP
   c. CPAP
   d. PSV
   e. AMV

3. Describe the hazards and side effects of each of the following modalities:

   a. SIMV
   b. PEEP
   c. CPAP
   d. PSV
   e. AMV

4. Describe and identify the pressure, volume, and flow waveforms for each of the following modalities:

   a. SIMV
   b. PEEP
   c. CPAP
   d. PSV
   e. AMV

5. List the four basic techniques that are used to assess the patient at the bedside.

6. Describe the factors that should be noted in each of the following areas of bedside patient assessment and be able to describe normal findings and interpret abnormal findings in each assessment area:

   a. Neurological assessment
   b. Pulmonary assessment
   c. Cardiovascular assessment

7. Define and explain the significance of the following terms:

a. Dyspnea
b. Cyanosis

8. Describe the indications and significance of data obtained from the following monitoring devices or lines:

   a. Oximeters
   b. CO_2 monitors
   c. Arterial lines
   d. Swan-Ganz lines
   e. CVP lines

9. Define the following terms associated with arterial blood gas analysis and interpretation:

   a. Acidosis
   b. Alkalosis
   c. Hypoxia
   d. Hyperoxia
   e. Acidemia
   f. Alkalemia
   g. Hypercapnia
   h. Hypocapnia
   i. Respiratory acidosis
   j. Respiratory alkalosis
   k. Hypoventilation
   l. Hyperventilation
   m. Hypoxemia
   n. Hyperoxemia
   o. Metabolic acidemia
   p. Metabolic alkalosis

10. Describe the physiological and clinical significance of each of the following arterial blood gas parameters:

    a. pH
    b. P_aCO_2
    c. P_aO_2

11. Be able to differentiate between the various types of compensation that can occur in arterial blood gases.

12. Given a set of arterial blood gases, be able to interpret them in terms of the acid-base and oxygenation status.

13. Describe the normal and primary abnormal findings associated with each of the following aspects of a chest x-ray.

    a. Bony structure
    b. Pleural space
    c. Heart
    d. Diaphragm
    e. Lung fields
    f. Airways

14. Describe the etiology, clinical manifestations, management, complications, and prognosis for each of the following diseases when mechanical ventilation is required:

    a. COPD
    b. Asthma
    c. ARDS
    d. Blunt-chest trauma
    e. Post-surgical recovery
    f. Myasthenia gravis
    g. Guillain-Barré
    h. Pulmonary edema

15. Describe various kinds of problems that can exacerbate respiratory failure and give examples for each general category.

16. Describe the general clinical picture of the patient in impending or frank respiratory failure and the kinds of variations that can occur.

17. How is respiratory failure definitively diagnosed?

18. Describe the indications and procedure for establishing an airway in the patient in respiratory failure.

19. Describe the procedure for establishing the initial ventilator parameters for the patient in respiratory failure.

20. Discuss the significance of the following factors in the ventilator patient for whom ventilator discontinuance or weaning is contemplated:

    a. Physiological preparation
    b. Cardiovascular status
    c. Nutrition
    d. Fatigue
    e. Fluid balance

21. Discuss the normal limits and usefulness of the following assessment procedures used to evaluate the ventilator patient's spontaneous ventilatory reserve:

    a. Arterial blood gases
    b. Alveolar-arterial oxygen gradient
    c. Peak inspiratory pressure
    d. Spontaneous minute ventilation
    e. Maximal voluntary ventilation
    f. Respiratory rate
    g. Vital capacity
    h. Forced expiratory volume in 1 second
    i. Spontaneous tidal volume
    j. V_D/V_T ratio
    k. Shunt fraction

22. Describe the application, advantages, and disadvantages of each of the following methods of ventilator discontinuance or weaning:

    a. Spontaneous breathing trials
    b. SIMV
    c. Pressure support
    d. AMV

INTRODUCTION

The Intensive Care Setting

The Intensive Care Unit (ICU) is an acute care environment designed to centralize the administration of critical health care to severely ill patients. Intensive care units are specialized today and a hospital may have the following areas:

a. Medical intensive care unit (MICU)
b. Surgical intensive care unit (SICU)
c. Cardiovascular surgical intensive care unit (CSICU)
d. Coronary care unit (CCU)
e. Respiratory intensive care unit (RICU)
f. Neonatal intensive care unit (NICU)
g. Pediatric intensive care unit (PICU)
h. Burn unit (BU)
i. Neurosurgical intensive care unit (NEICU)

Smaller hospitals may have only one or two intensive care areas that provide services to the total critical care population in the hospital. Larger hospitals usually have more specialized intensive care areas.

The goal of any intensive care unit is to maximize the level of care administered to the critically ill patient. This is accomplished in several ways. First, the intensive care area places the patient within close proximity to the staff. Second, ICU staff are trained and certified to administer the latest critical care procedures and medications. Third, the ICU incorporates the lastest monitoring and therapeutic equipment.

The Health Care Team

The competency of the individual care providers, and the level of teamwork determine the success of any intensive care area. The patient's care will be maximized by the administration of a comprehensive and integrated treatment plan rather than a more fragmented approach. This requires communication between the various individuals and departments involved in the care of the patients.

ICU health team members continually seek to maximize the patient's welfare. Such an attitude will guarantee the administration of the highest level of patient care.

VENTILATOR PROCEDURES

SIMV

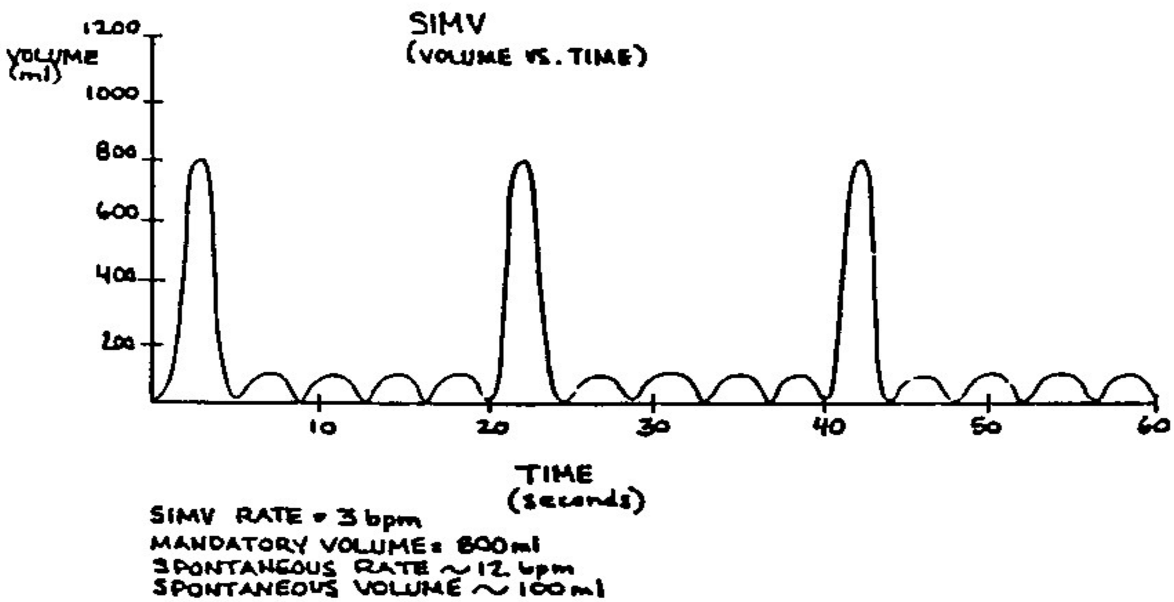
Synchronized Intermittent Mandatory Ventilation (SIMV) is a mechanical ventilator mode that allows the patient to breathe spontaneously through the ventilator circuit and at predetermined intervals, receive a volume from the ventilator (**Figure 1**).The inspired gas during the spontaneous breaths and the mandatory breaths should be identical with respect to:

1. Oxygen concentration
2. Humidity
3. Temperature

These criteria are easily met with newer ventilators.

Figure 1

SIMV



Most mechanical ventilators today incorporate the SIMV mode within the ventilator design, thus eliminating the need for auxiliary equipment. Most mechanical ventilators available today provide SIMV rather than IMV (Intermittent Mandatory Ventilation). The difference between these two similar modes is in the method the mandatory breath is delivered. In the IMV mode, the ventilator delivers the predetermined ventilator volume at a predetermined interval regardless of the status of the patient's spontaneous breath.

The ventilator might deliver the mandatory breath while the patient is at peak inspiration during his/her spontaneous breath, resulting in volume “stacking”. The SIMV mode eliminates this problem by synchronizing the delivery of the mandatory breath with the patient’s spontaneous breathing cycle. When the preset time interval has elapsed, the ventilator delivers the mandatory breath with the patient’s net inspiratory effort. Theoretically, SIMV is more appealing than IMV, although workers have shown no significant physiological difference between the two. Nonetheless, most ventilators now incorporate SIMV rather than IMV.

Originally, IMV was used to wean infants with IRDS from the ventilator. Later, it was found that the same procedure could be used to wean adult patients as well. SIMV is thought to be a better method of weaning, both physiologically and psychologically. When the patient is ready to be weaned, the SIMV rate is gradually decreased. As the SIMV rate is decreased, the patient is required to make up the difference in the total minute ventilation with an increase in his/her spontaneous breathing. The SIMV rate is systematically reduced until the patient is maintaining an adequate level of minute ventilation spontaneously. The patient should be allowed to stabilize at each decrease in the SIMV rate.

When a patient is on SIMV it is important that a record be kept distinguishing the ventilator rate, tidal volume, and minute ventilation from the patient’s spontaneous rate, tidal volume and minute ventilation. As the SIMV rate is reduced, the total minute ventilation should remain about the same. If the total minute ventilation decreases with a decrease in the SIMV rate, this indicates the patient’s spontaneous respiratory effort is inadequate with the decreased SIMV. The previous SIMV rate should be re-established.

PEEP

Positive End Expiratory Pressure (PEEP) is a ventilator modality whereby a constant positive pressure is applied to the lungs during the expiratory phase of the respiratory cycle (**Figure 2**). PEEP pressures of 5 to 15 cmH2O are commonly used. However, PEEP pressures up to 30 cmH_2O may be required in some patients. PEEP has been shown to improve oxygenation in patients who are severely hypoxic despite administration of high concentrations of oxygen. Although there are no hard and fast rules, it is generally felt that PEEP should be considered when the P_aO_2 remains below 50 mmHg on concentrations of oxygen greater than 50% in the patient on IPPV.

PEEP is applied in the patient with acute restrictive disease processes causing a reduction in the FRC. The reduction in the FRC is due to the collapse of the alveoli. This collapse may be due to intrabronchial, intra-alveolar, and/or interstitial fluid accumulation (**Figure 3**). This fluid accumulation prevents air from entering the alveoli and interferes with the production of surfactant within the alveoli, eventually leading to alveolar collapse. The loss of functional alveoli leads to physiologic shunting and hypoxemia. The hypoxemia is not corrected by administration of high concentrations of oxygen because the blood passing through the pulmonary capillaries comes into contact with alveoli that are no longer functioning as gas exchanging units. The loss of FRC also results in decreased compliance and increased work of breathing.

The initiation of PEEP will often improve oxygenation as a result of decreasing the amount of shunting. Because the FRC is improved, initiation of PEEP can also reduce the work of breathing by improving the lung compliance.

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Figure 2

PEEP

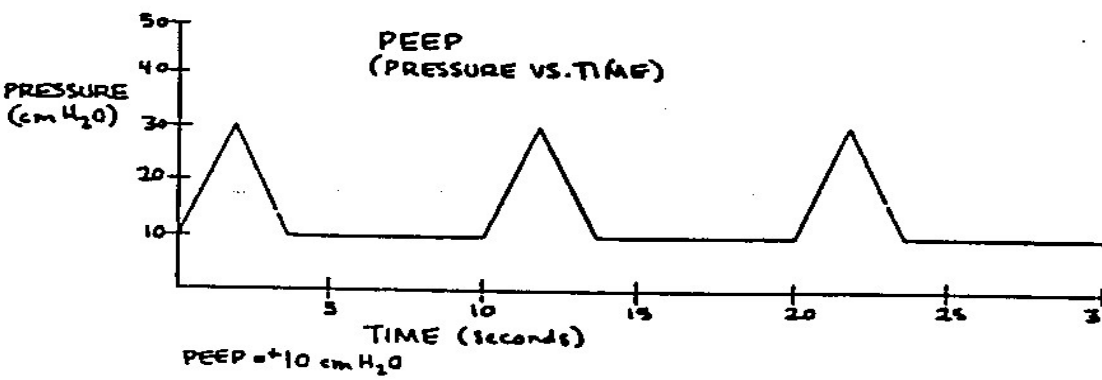
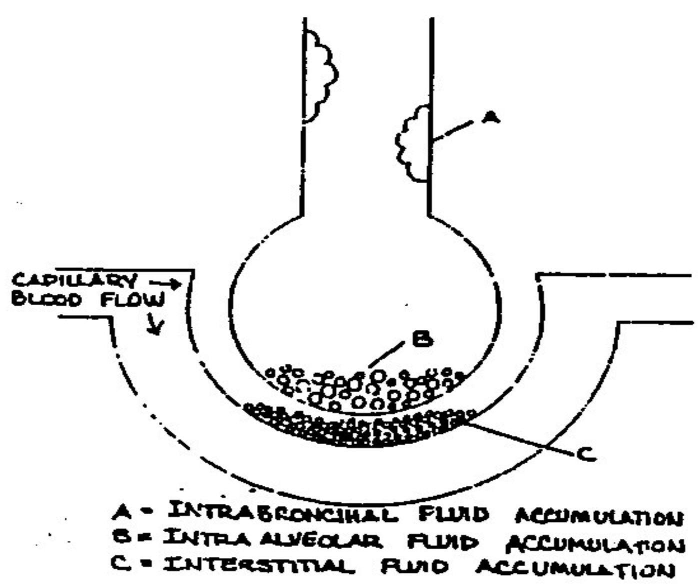


Figure 3

Fluid Accumulation in The Lungs



The most frequent undesired effect associated with the administration of PEEP is a reduction in the cardiac output. This is due to impeded venous return to the heart as a result of increased intrathoracic pressures. Since tissue oxygenation is determined by the amount of oxygen available in the blood and the amount of blood flow though the tissues, it follows that a reduction in the cardiac output will result in a decrease in tissue oxygenation. The administration of PEEP may improve the amount of oxygen in the blood but if the cardiac output is simultaneously reduced, an overall reduction in tissue oxygenation can occur. Therefore, the P_aO_2, by itself, is not an adequate means of evaluating the effectiveness of PEEP.

Ideally, the patient on PEEP should have a Swan-Ganz line in place allowing easy monitoring of the cardiac output in order to determine "optimal PEEP." If a Swan-Ganz line is not available then minimal monitoring should include:

1. Arterial Blood Gases
2. Pulse
3. Blood Pressure
4. Clinical Assessment of the patient.

An alternate method called "best PEEP" can be used to determine the maximal therapeutic level of PEEP in normovolemic patients without a Swan-Ganz line. It has been show that excessive PEEP pressures can actually decreas lung compliance, and that this reduction in lung compliance corresponds with a reduction in cardiac output. In clinical use, as PEEP is increased the effective static compliance is simultaneously measured. The initial increases in the PEEP level will normally be accompanied by an increase in the static effective compliance. PEEP is increased until there is no longer any improvement in the static effective compliance. At this point, any further increase in PEEP will result in cardiac impairment.

In patients exhibiting severe shunting, the goal should be to maintain a P_aO_2 of 60 mmHg. This is an adequate level of oxygenation in most patients. In patients with less severe shunting, it may be possible to maintain P_aO_2's in the 70 to 100 mmHg range, using lower levels of PEEP and F_IO_2.

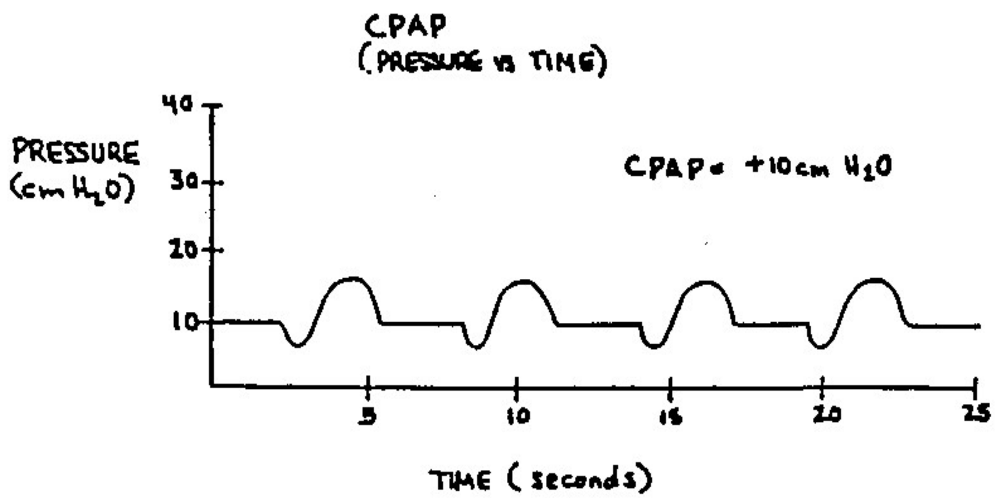
As the patient's oxygenation begins to improve, the F_IO_2 should be reduced first in 5 to 10% increments followed by arterial blood gases. When the F_IO_2 has been reduced to about 40%, the PEEP can be reduced in increments of 5 mmHg until the baseline pressure is reached.

CPAP

Continuous Positive Pressure Breathing (CPAP) is very similar to PEEP. The primary difference between these two modes is that PEEP is used in conjunction with IPPV whereas CPAP is used in conjunction with spontaneous breathing (**Figure 4**).

Figure 4

CPAP



CPAP is indicated in patients who are severely hypoxemic despite high concentrations of oxygen. They are able to maintain an adequate minute ventilation and P_aCO_2. The minute ventilation should be monitored on the CPAP patient since it is possible for their clinical situation to deteriorate resulting in hypercapnia and acidosis.

CPAP should be administered via a cuffed endotracheal or tracheostomy tube in adult patients to guarantee maintenance of the baseline pressure. The method for weaning CPAP is identical to PEEP. The F_IO_2 should be reduced first, followed by the CPAP.

Pressure Support Ventilation

Pressure Support Ventilation (PSV) is a ventilator mode that augments the spontaneous inspiratory effort using an operator-adjustable positive pressure applied only during the inspiratory phase of the ventilatory cycle. The gas flow and pressure are servo controlled as determined by the patient's breath-by-breath demands. PSV is an available modality on the Servo 900B and 900C, Bennett 7200, and Bear 5 ventilators.
PSV is a relatively new modality and the benefits and clinical applications have not been fully realized. Thus far, PSV is applied in one of two ways.

The first approach uses low pressures during PSV to simply overcome the resistance to gas flow through the endotracheal or tracheostomy tube. For example, a pressure gradient of about 4 cmH_2O is required to produce a flowrate of 40 lpm through a 7 mm endotracheal tube. Thus, a support pressure of 5-6 cmH_2O would counter the resistance of most endotracheal tubes, improving the patient's spontaneous ventilatory parameters (mainly the inspiratory flowrate and tidal volume) by reducing the work of breathing. The patients will also generally experience a subjective improvement in their spontaneous ventilatory capacity. Support pressures may be used in conjunction with SIMV or spontaneous breathing as the patient is being weaned from ventilator support.

The second approach uses pressures that are higher than needed to overcome the resistance to flow through the endotracheal or tracheostomy tube. A pressure of 10-15 cmH_2O might be used in this particular approach to further augment the patient's spontaneous respiratory effort. Again, the patient's spontaneous respiratory parameters will usually show noticeable improvement along with improvement in the patient's subjective evaluation of his/her respiratory capacity. This particular approach can also be used with SIMV or spontaneous breathing. It should be noted that support pressures up to 72 cmH_2O are available on the Bear 5 Ventilator. PSV will be a valuable mode in the management of ventilator patients with decreased spontaneous respiratory capacities.

Augmented Minute Ventilation

Augmented Minute Ventilation (AMV) is another recent ventilator mode that currently appears on the Bear 5 Ventilator. Essentially, AMV establishes a back-up minimum minute ventilation. The AMV mode operates just like SIMV except the average total exhaled minute volume (spontaneous minute volume and ventilator minute volume) must exceed the setting on the Minimum Minute Volume control. If the average total exhaled minute volume falls below the pre-set minimum minute volume because of a fall in the patient's spontaneous minute volume, the ventilator will deliver a pre-set volume at a computer-calculated back-up rate to maintain a minimum total minute volume.

As with PSV, AMV is a relatively new mode of ventilation. AMV appears to have application in weaning difficult patients from mechanical ventilation. The guaranteed minimum minute volume should be helpful in preventing dangerous fluctuations in the P_aCO_2 that can occur during SIMV and spontaneous breathing. AMV should prove to be a valuable ventilator mode in the future.

PATIENT EVALUATION

Effective ventilator management requires intelligent and pertinent patient evaluation. Ventilator monitoring is important but is no substitute for patient assessment. The therapist assigned to a ventilator patient is responsible for maintaining adequate ventilation and oxygenation of the patient. This certainly involves more than periodic ventilator checks.

Bedside Assessment Procedures

Adequate management of the continuous mechanical ventilator patient includes proper bedside monitoring. The respiratory care practitioner must learn to work in a continuous state of evaluative awareness. It is only in this mode that the more subtle patient changes can be perceived. A thorough initial evaluation is necessary to establish a clinical baseline. Thereafter, frequent evaluation is necessary to detect any changes in the patient's condition.

There are four basic techniques involved in bedside patient assessment:

1. Observation
2. Palpation
3. Percussion
4. Auscultation

These techniques are used to evaluate the various aspects of a patient's condition and are referred to in the following discussion of a patient assessment procedures.

Neurological Assessment

Hypoxemia and hypercapnia can affect the mental status of the patient. Moderate hypoxemia produces dyspnea, which is often associated with anxiety. The patient with severe hypoxemia may become disoriented, lethargic and even comatose. These changes can occur suddenly or over a long period of time. The patient's neurological status can often be quickly determined by his/her ability to respond to verbal and painful stimuli.

Pulmonary Assessment

<u>Respiratory Pattern</u>

Observation of the respiratory pattern should include several items. The rate and depth of breathing should be noted. Although the rate can be evaluated objectively, the depth of respiration is a rather subjective evaluation and should be used only as a rough guide for assessing the patient's volume exchange. The I:E ratio should be noted. In the normal patient the I:E ratio is about 1:2. In obstructive pulmonary disease, the expiratory time is significantly prolonged.

The chest should be observed to identify the presence or absence of accessory muscle use. While observing the chest, the respiratory care practitioner should also watch for bilateral chest expansion. Decreased chest expansion on one side may indicate atelectasis or a pneumothorax. In chest trauma, the chest should be examined for "flailing."

Chest Auscultation

Chest auscultation should identify:

a. Intensity (loudness)
b. Quality
c. Pitch of breath sounds

In the normal patient, the inspiratory phase is easily heard while the expiratory phase is less audible and is not heard throughout exhalation. Breath sounds may be described as having a vesicular, bronchial or adventitious quality. Adventitious sounds include rales, rhonchi, wheezing and pleural rubs. Vesicular breath sounds are associated with normal lungs and can be heard over most of the chest. The inspiratory phase is louder, higher-pitched, and of longer duration than the expiratory phase. There is no pause between the inspiratory and expiratory phase. Bronchial breath sounds are loud and harsh and have a tubular quality. The expiratory phase is usually longer and louder than the inspiratory phase and a pause usually occurs between inspiration and expiration. Rales are the most common adventitious breath sound. Rales are produced by the passage of air though small airways containing fluid. They can be described as crackling sounds. Rhonchi occur in airways where accumulation of fluid or secretions has occurred. Rhonchi can vary from high-pitched to low-pitched bubbling sounds depending on the size of the airway and are usually associated with expiration. Rhonchi can often be cleared by suctioning or coughing. Wheezing is actually high-pitched rhonchi. The sound is produced by air passing through restricted airways as in asthma. Wheezing is usually more pronounced on expiration and is often corrected by administration of a nebulized bronchodilator. Pleural rubs are creaking, leathery sounds that occur at the end of inspiration and beginning of expiration. Pleural rubs are due to inflammation of the visceral and parietal pleura.

The distribution of the intensity of breath sounds should be noted both vertically and horizontally. Auscultating vertically, the breath sounds will normally be less intense in the bases. Auscultating horizontally, the intensity of the breath sounds in the right and left lungs should be equal. The horizontal distribution of breath sounds is particularly important in assessing the position of an endotracheal tube in the intubated patient. Finally, the adventitious breath sounds should be identified and described, noting their location and frequency.

Sputum

The patient's sputum can reveal significant information about his/her lung disease. The following qualities should be noted:

1. Volume
2. Consistency
3. Purulence
4. Color
5. Odor

6. Presence of blood

Patients with COPD will have large amounts of sputum production. This is particularly true of the patient with bronchiectasis or chronic bronchitis. They may produce more than 100 ml of sputum in a 24 hour period. Other lung diseases associated with copious amounts of sputum production are abscesses, cystic fibrosis and lung cancer.

An increase in clear, mucoid secretions is due to an inflammatory response to some allergen in the airways, typical in the asthmatic patient. Purulent secretions indicate the presence of pathogenic bacteria and infection. Purulent secretions are common in pneumonia, acute bronchitis, and aspiration pneumonia. Purulent secretions are usually either yellow or green. The presence of a fetid sputum may indicate a lung abscess, bronchiectasis, cystic fibrosis or aspiration pneumonia.

The presence of blood should be distinguished into two categories: 1) bright red and 2) rusty. Bright red blood in the sputum is associated with tuberculosis, carcinoma of the lung and bronchiectasis. Rusty or darker blood in the sputum is associated with some pneumonias. Blood in the sputum may be associated with bleeding in the nasopharynx or other parts of the upper airways. It is also possible for blood from the stomach and esophagus to be expectorated in the sputum. Thus, the presence of blood in the sputum does not necessarily indicate an underlying pulmonary condition. If there is bright red blood in the sputum, the source of the bleeding should be identified and corrected. Patients with severe bleeding may require blood transfusions.

Dyspnea

Dyspnea is a subjective perception. The patient senses difficulty in breathing. As such, dyspnea cannot be observed or quantified. It can only be reported that the patient feels dyspneic. Most dyspneic patients will have an increased work of breathing. It can be assessed by observing their respiratory pattern, respiratory rate and level of anxiety. Arterial blood gases will often confirm the presence of inadequate ventilation or oxygenation in the dyspneic patient; however, it is possible for a patient to have normal arterial blood gases and still complain of dyspnea. The exact cause of dyspnea in such cases is unknown.

Other Observations

There are other clinical signs that can also yield information about the pulmonary status of a patient. Cyanosis, a bluish discoloration of the skin and mucous membranes, may indicate a reduced oxygen level in the blood; however, it is not a reliable indicator of the patient's oxygenation status for several reasons. First, cyanosis does not normally occur unless the P_aO_2 is below 50 mmHg. The patient can be moderately hypoxemic and not be cyanotic. Second, the amount of oxygen desaturation that must occur before cyanosis appears is dependent upon the amount of hemoglobin. Polycythemic patients will show cyanosis at higher levels of saturation whereas the anemic patient can be severely desaturated and show no cyanosis. Third, cyanosis may occur in extremities as a result of either a cold environment or effects of the nervous system. Lastly cyanosis is not easily detected in patients with darker skin pigmentation.

Cardiovascular Assessment

Bedside evaluation of the cardiovascular system should include an assessment of the blood pressure, pulse and perfusion. The state of the patient's perfusion can best be estimated by noting the color and texture of the skin and the capillary refill. Adequate urinary output and normal mentation also indicate adequate tissue perfusion and oxygenation.

Arterial Line

The arterial line is attached to a pressure transducer that produces an arterial pressure wave form on an oscilloscope. Besides the systolic and diastolic pressures, mean arterial pressure is also given. The arterial line also allows for frequent blood gas analysis and withdrawal of blood for other laboratory tests.

CVP Line

The central venous pressure (CVP) is useful in assessing the relationship between the pumping action of the heart and the circulatory blood volume. The CVP reflects the systemic venous return, which is a primary determination of the left ventricular output. The CVP is obtained by placing a catheter at the junction of the superior vena cava and right atrium, which is attached to a water manometer and read in cmH_2O. The normal value is 5-15 cmH_2O. Serial changes in the CVP may indicate hypovolemia. Coinciding with an improvement in the patient's condition, a declining CVP may indicate an improvement in the cardiac output. On the other hand, a high CVP may indicate fluid overload or cardiac failure, caused by an inability of the heart to pump blood returning to it. CVP data is useful only if the right and left heart are functioning equally. Often the left heart is incapable of maintaining an adequate output. If this situation is suspected in the critically ill patient, a pulmonary artery catheter should be inserted to monitor left heart function.

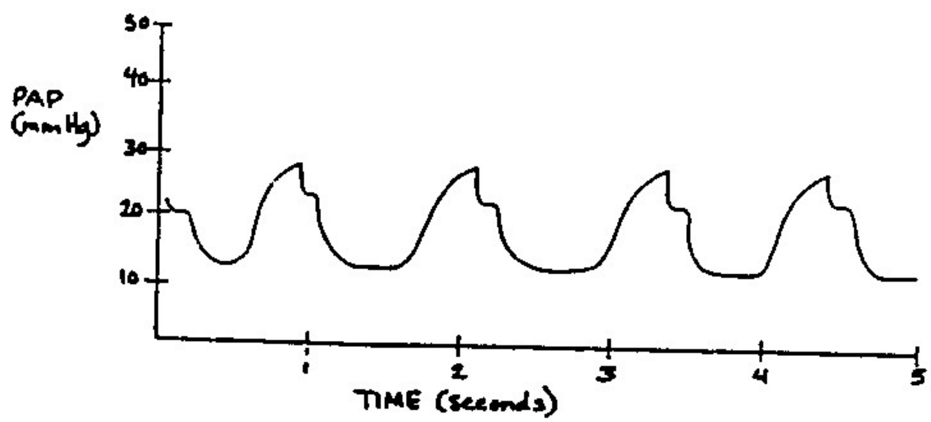
Pulmonary Artery Catheter

The Swan-Ganz line provides a means of measuring the pulmonary artery pressure (PAP), wedge pressure, and cardiac output. The simplest catheter has two lumens; one is in contact with the blood and the other is connected to a balloon located near the distal end of the catheter. The catheter is introduced into the venous system via a cut-down at the antecubital fossa, percutaneously at the subclavian or jugular veins, or via the femoral vein. The catheter is initially advanced with the balloon deflated until the tip of the catheter is within the thoracic cavity. The balloon is then inflated and advanced further. The balloon acts like an umbrella with the blood flow advancing the catheter through the heart and out into the pulmonary artery. With the catheter in proper

position in the pulmonary artery, a pressure wave form similar to the one shown in **Figure 5** will appear. The normal PAP is about 25/10 mmHg. In addition to the PAP, the pulmonary wedge pressure (PWP) can be obtained by inflating the balloon on the distal end of the Swan-Ganz catheter. The PWP gives an indirect assessment of the left atrial pressure. The normal PWP is 4-12 mmHg (**Figure 6**). An increase in the PWP indicates an inability of the left heart to pump the blood returning to it (left ventricular failure). Lastly, the Swan-Ganz catheter can be used to determine the cardiac output. This is most easily done by using a four lumen catheter rather than the simple two lumen catheter. The third lumen has a hole approximately 30 cm proximal to the distal end of the catheter, while the fourth lumen is attached to a thermistor, which is located at the distal end of the catheter. A cold solution is injected through the hole of the third lumen passing into the right atrium or superior vena cava. As the fluid travels towards the thermistor, it is heated by the surrounding blood. The thermistor measures the final temperature of the fluid, and a computer calculates the cardiac output based on the difference in the initial and final temperature.

Figure 5

Pulmonary Artery Pressure



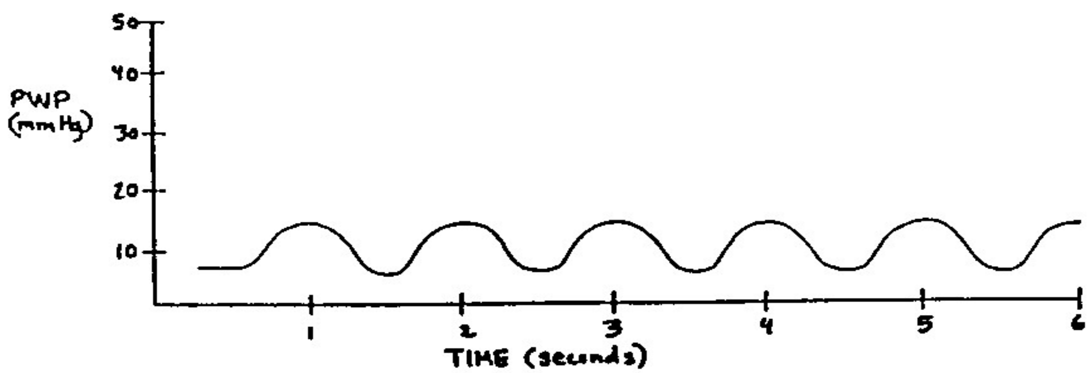
BEDSIDE MONOTORING DEVICES

Oximeters

Continuous non-invasive monitoring of oxygen saturation has been made available with the introduction of several transcutaneous oximetry devices. These devices are simple to operate and give reliable data. They are useful in monitoring patients with inadequate and/ or changing oxygenation.

Figure 6

Pulmonary Wedge Pressure



Oximeters use either the polarographic or spectrophotometric principles of operation to indirectly determine the saturation of arterial blood. The polarographic units have a Clark electrode that senses the P_aO_2 on the surface of the skin. Heat is applied to the electrode to cause hyperemia of the skin so that the surface PO_2 more closely approximates the P_aO_2. The spectrophotometric units use the principle of light absorption to approximate the amount of oxyhemoglobin in the blood. A microprocessor analyzes the absorption of different wave lengths of light and calculates the S_aO_2.

Readings from the oximeter should be correlated initially and periodically with arterial blood gases. Usually the saturation given by the oximeter will be within 2-4% of the S_aO_2 obtained from the arterial blood gas. Significant changes in the saturation reported by the oximeter will usually indicate a corresponding change in the arterial saturation of oxygen.

Bedside oximetry is indicated in patients with decreased or fluctuating oxygenation. Oximetry can also help reduce the number of arterial blood gases needed to effectively monitor the oxygenation status.

CO_2 Monitors

Continuous non-invasive bedside monitoring of carbon dioxide can be just as essential, perhaps more so, as continuous non-invasive monitoring of the oxygen saturation. This is true because of the relationship between the P_aCO_2 and the pH. Rapid and significant changes in the P_aCO_2 result in immediate changes in the pH, which can produce a life-threatening acidosis, or alkalosis in the ventilator patient.

Gaseous carbon dioxide can be measured by infrared irradiation, thermal conductivity, the Severinghaus electrode, or mass spectrometry. Most bedside CO_2 monitoring equipment operates on the infrared irradiation principle in which different gases produce different absorption spectra. The infrared light is absorbed by carbon dioxide and water vapor but not by other atmospheric gases such as oxygen and nitrogen or trace gases such as helium and argon. The device measures the amount of infrared light absorbed by the carbon dioxide present in the gas sample. These devices have a relatively rapid response time and can give reliable data. The small diameter sampling line must be changed routinely or flushed with a dry gas to prevent accumulation of condensation and secretions which block the flow of gas into the device, resulting in false low readings.

Continuous non-invasive monitoring of CO_2 is particularly indicated in the unstable ventilator patient with hypercapnia. Periodic arterial blood gases may not detect a sudden and progressive increase in the P_aCO_2. Continuous monitoring of the carbon dioxide also provides a better means for evaluating changes made in the ventilator minute ventilation and may be helpful in monitoring the patient being weaned from mechanical ventilation. Finally, it reduces the need for arterial blood gases, reducing the discomfort, potential hazards, and cost to the patient.

The CO_2 monitor should be calibrated with a known gas sample (usually 5% carbon dioxide and 95% nitrogen). The sampling line should then be attached as close as possible to the patient's proximal airway. It is important that an arterial blood gas sample be taken to correlate the readings on the CO_2 monitor with the actual P_aCO_2. The P_aCO_2 and the CO_2 given on the CO_2 monitor should be within 5 mmHg. If not, the CO_2 monitor should be recalibrated and checked for proper functioning. A space on the ventilator flow sheet should be provided for recording values.

LABORATORY ASSESSMENT PROCEDURES

Arterial Blood Gas Analysis

In the management of any respiratory patient, arterial blood gases are essential for assessing the patient's clinical condition and for proper management of the patient. This is particularly true for patients receiving continuous mechanical ventilation.

To help standardize the terminology used to describe blood gas values, various terms commonly used in the critical care setting are defined in **Table 1**.

The P_aO_2 is a direct measurement of the amount of dissolved oxygen in the arterial blood. Although a good indicator of the pulmonary status of the patient, the P_aO_2 by itself does not provide an adequate picture of tissue oxygenation. Two other factors that determine the adequacy of tissue oxygenation are the C_aO_2 and the cardiac output. A reduction in either of these two parameters will compromise tissue oxygenation. Thus, it is possible for a patient to have a normal P_aO_2 and be hypoxic. Hypoxia is a term describing the patient's tissue oxygenation, inferred from the assimilation of laboratory and clinical assessment information.

The P_aCO_2 is the easiest way and most direct means of assessing the adequacy of alveolar ventilation. Since the P_aCO_2 is inversely related to the alveolar ventilation, an increase in the P_aCO_2 will reflect a decrease in the alveolar ventilation, as shown below.

$$P_aCO_2 \cong \frac{CO_2\ \text{Production}}{\text{Alveolar ventilation}}$$

The arterial pH is a measurement of the hydrogen ion concentration in the arterial blood. The pH has a metabolic and a respiratory component. The P_aCO_2 (respiratory component) is inversely related to the pH, whereas the HCO_3 (metabolic component) is directly related to the pH as shown below.

$$\text{pH} \cong \frac{HCO_3}{\text{Alveolar ventilation}}$$

Thus, an overall evaluation and description of the pH requires an examination of the P_aCO_2 and HCO_3. A summary of the interaction between the pH, P_aCO_2, and HCO_3 and a description of the various combinations is provided in **Table 2**.

In order to differentiate respiratory compensation from metabolic compensation it is necessary to know something about the patient's clinical history. If a patient has a history of COPD, an increased P_aCO_2 accompanied with an increased HCO_3 and a normal pH, represents a compensated respiratory acidosis, not a compensated metabolic alkalosis. Also, a compensated respiratory acidosis will show a pH that is slightly acidotic (7.30-7.40). A compensated metabolic alkalosis will show a pH that is slightly alkalotic (7.40-7.50). Also, a compensated respiratory acidosis is much more common than a compensated metabolic alkalosis.

The patient's clinical history needs to be known to differentiate between a compensated metabolic acidosis and a compensated respiratory alkalosis. Compensated metabolic acidosis is more common and it is not uncommon for the respiratory component to slightly over-compensate for a metabolic acidosis resulting in a slightly alkalotic pH.

Chest X-Rays

The respiratory care practitioner working with critically ill patients should be familiar with the basics of chest roentgenographic evaluation. It is important that a chest x-ray be examined in a systematic fashion. The following characteristics should be examined:

Table 1

TERMINOLOGY FOR DESCRIBING CLINICAL AND BLOOD GAS ABNORMALITIES

I. Terms Describing Patient's General Condition

Term	Definition
Acidosis	A condition characterized by a base deficit.
Alkalosis	A condition characterized by a base excess.
Hypoxia	A condition in which the tissue oxygenation is inadequate for proper cellular metabolism.
Hyperoxia	A condition in which the tissue oxygenation is greater than what is required for cellular metabolism.

II. Terms Describing the pH

Term	pH value
Acidemia	An arterial pH below 7.30
Alkalemia	An arterial pH above 7.50

III. Terms Describing P_aCO_2

Term	P_aCO_2 Value
Hypercapnia	A P_aCO_2 above 50 mmHg.
Hypocapnia	A P_aCO_2 below 30 mmHg.
Respiratory Acidosis	A P_aCO_2 greater than 50 mmHg.
Respiratory Alkalosis	A P_aCO_2 less than 30 mmHg.
Hypoventilation	A P_aCO_2 greater than 50 mmHg.
Hyperventilation	A P_aCO_2 less than 30 mmHg.

IV. Terms Describing the P_aO_2

Term	P_aO_2 Value

Hypoxemia	A P_aO_2 below 60 mmHg (in the patient below 60 years of age).
Hyperoxemia	A P_aO_2 greater than 110 mmHg.

V. Terms Describing the HCO_3

Term	HCO_3 Value
Metabolic Acidemia	An HCO_3 less than 20 mEq/liter.
Metabolic Alkalemia	An HCO_3 greater than 30 mEq/liter.

Table 2

CLASSIFICATION OF THE ACID-BASE STATUS

pH	P_aCO_2	HCO_3	Description
→	→	→	Normal Acid-Base Status
↓	↑	→	Respiratory Acidosis
↑	↓	→	Respiratory Alkalosis
↓	→	↓	Metabolic Acidosis
↑	→	↑	Metabolic Alkalosis
→	↓	↑	Compensated Respiratory Acidosis or Compensated Metabolic Alkalosis
→	↑	↓	Compensated Metabolic Acidosis or Compensated Respiratory Alkalosis

Bony Structure

The rib cage should be examined for abnormalities. The ribs should be equidistant. If the ribs on one side of the chest are compressed, this could be an indication of atelectasis on that side. Ribs that are elevated on both sides indicate either acute or chronic airway obstruction. It is important to realize that the position of the patient can affect the appearance of the rib cage and give false impressions. Any fractures in the ribs should be identified.

Pleural Space

Normally the pleural space is only a "potential" space between the visceral and parietal pleura. The entire lung border should be checked for abnormalities such as: 1) the presence of air or fluid, 2) the presence of masses 3) the blunting of the costophrenic angle due to fluid accumulation.

Heart

The size of the heart should be noted since this can be an indicator of congestive heart failure. Normally, the width of the heart should be no greater than one half the distance across the lungs at the level of the diaphragms. The heart shadow looks slightly enlarged in the AP chest x-ray due to the distance of the heart from the film.

Diaphragm

A chest x-ray is normally taken with the patient in full inspiration. Thus, the diaphragms should be lower and appear flattened. The right diaphragm should be slightly higher than the left. A diaphragm that is abnormally elevated could indicate atelectasis. Abnormally flat hemi-diaphragms could indicate acute or chronic airway obstruction.

Lung Fields

The lung fields should be examined for overall translucency. In the normal lung, the practitioner should see lung markings throughout both lung fields. The lung fields should then be examined for any localized areas of increased or decreased translucency. Areas of increased translucency represent an increase in the ratio of the air to tissue as seen in pneumothoraces, blebs, asthma and COPD. The presence of fluid in the lungs will decrease the translucency resulting in a white appearance on the x-ray film. Atelectasis will also produce a less translucent picture since the ratio of air to tissue is decreased.

Airways

The position of the trachea should be noted. The trachea should be midline on the chest x-ray. One can determine the position of the trachea by determining its relationship to the vertebral spines. The vertebral spines should go through the middle of the trachea. A tracheal deviation could be due to improper position of the patient during the x-ray. This can be determined by noticing the position of the clavicles. Assuming the patient was in proper position, a deviation in the trachea could be the result of several things. Atelectasis tends to draw the trachea toward the affected side. Collapsed lung segments or lobes also draw the trachea toward the affected side. The respiratory care practitioner should be able to identify the carina to determine the position of endotracheal tubes. The tip of the endotracheal tube should be at least 2-3 cm above the carina.

SITUATIONS REQUIRING VENTILATOR ASSISTANCE

COPD

Etiology

The term chronic obstructive pulmonary disease (COPD) describes a class of obstructive diseases that usually appear in some combination.

Some believe the term COPD is inappropriately applied to what is actually several distinct respiratory diseases. Although it is true that COPD is a broad term describing a process involving several specific respiratory diseases, the term is not entirely inappropriate since most patients exhibiting chronic airway obstruction have a combination of respiratory diseases. The obstructive pulmonary diseases that are typically involved include:

1. Chronic Bronchitis
2. Emphysema
3. Bronchiectasis
4. Asthma

COPD is usually a combination of chronic bronchitis and emphysema with a variable component of asthma. The patient with COPD may also have right heart failure (cor pulmonale) and left heart failure. The asthmatic patient may develop COPD. In this type of patient, bronchodilator therapy is beneficial in treating the asthmatic component of the disease; however, as the emphysema and chronic bronchitis progress, the effect of bronchodilator therapy decreases.

Clinical Manifestations

The lungs in the patient with advanced COPD are so severely damaged that the capacity of the respiratory system becomes extremely compromised. Ordinarily, the COPD patient is able to maintain a baseline level of ventilation and oxygenation.

The COPD patient presents with a history of chronic cough, sputum production and varying degrees of wheezing and rhonchi. The chest is hyperexpanded (barrel chest) and accessory muscles used. Expiration is prolonged, particularly in the patient having emphysematous changes in the lungs. Endotracheal is usually present. The patient may be cyanotic, depending on the exact nature of his/her disease. Patients who have a larger component of chronic bronchitis are usually cyanotic, whereas patients having primarily emphysema are not. The chest x-ray reveals hyperexpansion with flattened diaphragms, elevated ribs, and generalized translucency. Arterial blood gases reveal a compensated respiratory acidosis accompanied by hypoxemia on room air.

The COPD patient may develop respiratory failure secondary to infection, bronchospasm or heart failure. The patient's sensorium will vary depending on the severity of the acidosis and hypoxia. In the early stage of acute exacerbation, the patient will be anxious and sometimes confused; however, as tissue oxygenation is further impaired, he/she may become lethargic and comatose. At this point, immediate medical intervention is indicated. Arterial blood gases will reveal a P_aCO_2 well above the patient's baseline (70-100 mmHg) accompanied by an acidosis (pH below 7.25) and hypoxemia on supplemental oxygen (P_aCO_2 below 50 mmHg).

Management

The COPD patient in respiratory failure should be intubated and placed on continuous mechanical ventilation. An acidosis that is primarily due to hypercapnia should not be corrected with sodium bicarbonate. Instead, the patient's CO_2 should be gradually reduced to improve the pH. If a substantial metabolic acidosis is also present, it may be desirable to correct some of the base deficit with sodium bicarbonate. It is also important to not over ventilate the patient. The ventilator should be adjusted to achieve and then maintain the patient's baseline acid-base status. It is very helpful at this point to know something about the patient's history. Knowledge of the patient's baseline arterial blood gas values and pulmonary function studies are extremely valuable in establishing goals for ventilation and weaning from the ventilator. The goal of mechanical ventilation should be to bring the arterial blood gas values back into range of the patient's baseline values. This will mean keeping the pH at 7.40 or below and the P_aCO_2 above 50 mmHg. Likewise, it is not necessary to achieve P_aO_2's much greater than 60 mmHg.

Nasal intubation is preferred since the COPD patient usually requires several days of mechanical ventilation. Tracheostomy should be considered in patients who will require ventilation for more than 10-14 days.

Severe wheezing is often present. The wheezing may be the result of pulmonary edema secondary to cor pulmonale and congestive heart failure. Digitalis may be used to increase the contractility of the heart, reduce the heart rate, and suppress ventricular arrhythmias. Diuretics help remove the accumulation of extravascular fluid in the lungs and reduce wheezing. Bronchodilator therapy may also be used as an adjunct to help correct bronchoconstriction. Often, wheezing in the COPD patient is due to the presence of allergens or infection in the airways. Treatment consists primarily of bronchodilator therapy including IV aminophylline and nebulized bronchodilators. Respiratory tract infections should be treated with an appropriate antibiotic. It is important that the primary cause of the wheezing be determined so that proper medical treatment can be implemented.

Weaning from the ventilator should not be attempted until the patient's respiratory and cardiovascular systems are stabilized. Methods and criteria for weaning are discussed in a subsequent section.

Complications

Nosocomial infections can be devastating to the COPD patient receiving mechanical ventilation. Airway management must incorporate the utmost attention to adequate secretion evacuation employing sterile equipment and procedures. The ventilator circuit should be changed at least every day. Trach care should be done every 8 hours adhering to strict sterile techniques. And lastly, thorough hand washing goes a long way in reducing the incidence of nosocomial infections.

The emphysematous patient may occasionally develop pneumothoraces from the application of positive pressure to bullous areas of the lungs. The incidence of pneumothoraces is related to the severity and number of bullae in the lungs and the airway pressure generated by the ventilator. The use of large tidal volumes and high inspiratory flowrates contribute to the incidence of pneumothoraces in the emphysematous patient and should be avoided if possible. A sudden deterioration in the patient's condition coinciding with localized absence of breath sounds, increased work of breathing, increased peak airway pressures, anxiety and/or tachycardia suggest a pneumothorax. A STAT chest x-ray should be obtained to confirm any suspicion. Substantial pneumothoraces should be treated with chest tubes.

Since COPD patients often require mechanical ventilation for extended periods of time, they may experience a variety of emotional reactions including anxiety, fear, despondency, bitterness and rejection. The respiratory care practitioner often sees the patient more often than the other members of the medical team. As a result, the practitioner can become a key person in assuring that the patient's emotional needs are met. Communication is a very important factor. This includes listening to the patient. Although the patient's ability to communicate is impaired because of the artificial airway, one should not assume that he/she cannot communicate. The patient can communicate if the practitioner is willing to take the time to work with him/her.

Lastly, the COPD patient can become ventilator-dependent. Although ventilator dependence can occur, there are measures that can be taken to reduce this occurance. As soon as mechanical ventilation is initiated, the first goal should be stabilization of the patient. Weaning should not be attempted until this is achieved. Very often, early attempts at weaning ultimately result in further deterioration of the patient's condition, thus prolonging the course of mechanical ventilation. When the patient is ready to be weaned, the process should be tailored to his/her condition and ability. In general, the length of time required to wean a patient will correlate with the length of time on the ventilator. This is why it is important to stabilize the patient as quickly as possible. As the time on the ventilator lengthens, the weaning process becomes more difficult. Finally, the patient's nutritional needs must be met to avoid weight loss and muscle weakness. Debilitated patients will not tolerate weaning from the ventilator.

Prognosis

The prognosis for the COPD patient who requires mechanical ventilation is quite variable and depends on a number of factors. Patients who are older and have long-standing COPD with several previous occurances of acute exacerbation requiring mechanical ventilation, generally have a poor prognosis. Other accompanying medical problems such as congestive heart failure, diabetes, myocardial infarctions, and hypertension worsen the prognosis in these patients. Acute exacerbation in the COPD patient can be reduced by means of an aggressive and continuing rehabilitation program that includes:

1. Instruction about their disease
2. Pharmacological therapy
3. Good hygiene
4. Breathing instruction
5. Exercise
6. Improved Nutrition
7. Periodic evaluation and adjustment of their treatment regime

Chronic bronchitis

Is characterized by a chronic cough with excessive sputum production which persists for more than three months out of the year for two or more consecutive years. There is hypertrophy of the mucous glands and goblet cells resulting in increased mucous production. This is accompanied by decreased ciliary activity. Both of these problems are highly correlated with cigarette smoking. This term "blue bloater" describes the patient who primarily has chronic bronchitis.

Emphysema

Is a nonreversible pulmonary disease characterized by destruction of pulmonary septal walls and connective tissue beyond the terminal bronchioles. This causes a loss of lung elastic recoil and alveolar hyperdistention. Emphysema occurs commonly with chronic bronchitis but can also occur alone. The term "pink puffer" describes the patient who primarily has emphysema.

Bronchiectasis

Is a pulmonary disease exhibiting abnormal dilation and distortion of the bronchi and/ or bronchioles. The etiology of bronchiectasis is debated though it appears that any lesion which narrows a bronchiole lumen may produce bronchiectasis. The disease is associated with increased cough and production of purulent sputum.

Asthma

Is an intermittent, reversible pulmonary disease that may occur in children and adults. The exact etiology of asthma is unknown though it is precipitated by factors such as:

1. Infections
2. Emotional stress
3. Allergens
4. Cold weather
5. Exercise

The primary symptoms of asthma are wheezing and dyspnea. Asthma can usually be managed with long-term bronchodilators and steroids. Sudden attacks can usually be reversed with bronchodilator administration.

Asthma

Etiology

The exact etiology of asthma is unknown although it is well known that many factors mentioned earlier can precipitate bronchospasm. Airway obstruction occurs in both the large and small airways. Obstruction in the larger airways is responsible for the increased airway resistance.

Status asthmaticus is an acute exacerbation that is non-responsive to bronchodilator therapy. Status asthmaticus is a life-threatening situation that requires immediate medical intervention.

Clinical Manifestations

The cardinal sign of asthma is wheezing associated with a lengthening of expiration. The patient has a productive cough with a thick, clear, mucoid sputum. The patient in moderate distress will complain of dyspnea and will appear to have respiratory difficulty as evidenced by tachypnea, use of accessory muscles, anxiety, cyanosis, and audible wheezing. The chest will distended and will be hyperresonant to percussion. The chest x-ray will confirm hyperinflation of the chest with flattened diaphragms, elevated ribs and generalized increased translucency of the lung fields. In mild asthma the blood gases will reveal a normal acid-base status with a mild hypoxemia on room air due to ventilation/perfusion mismatch. As the asthma progresses, the patient will begin to retain CO_2 and will be hypoxic on supplemental oxygen. Even small increases in the P_aCO_2 are significant in the asthma patient and indicate serious obstruction. At this point, the patient may become fatigued and obtunded. The breath sounds may diminish. So little air may be moved by the patient that wheezing and rales may become inaudible. Cyanosis may become marked. At this point the patient may not respond to bronchodilators and respiratory failure may be impending. Prompt and appropriate medical intervention is essential.

Management

The asthmatic patient is normally managed on a regimen that includes aerosolized or oral bronchodilators, decongestants, steroids and agents that block the allergic response. These drugs coupled with a reduction of environmental irritants and emotional stress are effective in the long-term management of the asthmatic patient. However, the patient who comes to the emergency room in status asthmaticus will not respond to bronchodilators and can progress into respiratory failure. The patient who exhibits a P_aCO_2 greater than 55 mmHg and a P_aO_2 less than 60 mmHg on 50% oxygen and does not respond to bronchodilator therapy should be intubated and placed on continuous mechanical ventilation. He/she should be sedated with morphine to reduce anxiety and improve the efficiency of ventilation.

Some have found the administration of IV Isoproterenol to be more effective than aminophylline due to its short half life (5 minutes compared with 3 hours for aminophylline). Due to the potential adverse cardiovascular effects of Isoproterenol, precise titration is required and the drug should not be given to patients who have underlying cardiac complications. Frequent aerosolized bronchodilator treatments and chest physiotherapy should be administered to improve ventilation and mobilization of secretions. The patient should require no more than 3-4 days of mechanical ventilation. Improved arterial blood gases coupled with improved breath sounds indicate that mechanical ventilation can be terminated and the patient extubated. The adequacy of the patient's spontaneous ventilatory parameters should be verified before this is done.

Complications

Though status asthmaticus is a life-threatening situation, very few complications are associated with the disease. Patients may develop secondary viral or bacterial infections. In fact, viral infections are often responsible for precipitating asthma. Bacterial infections should be treated with an appropriate antibiotic. Prophylactic administration of antibiotics when a viral infection is suspected is not indicated. Occasionally, the patient in status asthmaticus may develop a spontaneous pneumothorax due to air-trapping and hyperinflation of alveoli.

Prognosis

The prognosis for the patient in status asthmaticus is very good. With proper medical intervention, status asthmaticus is reversible in most cases. This is particularly true in children.

ARDS

Etiology

Adult Respiratory Distress Syndrome (ARDS) is the name given to the pulmonary condition associated with a wide variety of causal factors. Some of the factors that are correlated with the development of ARDS include:

1. Oxygen toxicity
2. Near drowning
3. Severe pneumonitis
4. Trauma
5. Post-cardiopulmonary bypass
6. Shock

This list is not exhaustive but illustrates the wide range of disposing factors. The onset of ARDS usually occurs within 12 to 48 hours of the initial injury.

Clinical Manifestations

The major pathological problems include:

1. Increased permeability of the alveolarcapillary membrane, which allows fluid, proteins and blood elements to leak into the alveolar space

2. Increased pulmonary vascular resistance, which increases the work load on the right heart

3. Reduction in surfactant production by Type II cells leading to alveolar collapse, shunting, and decreased compliance

The initial phase of the disease process may be associated with mild dyspnea, increased work of breathing, tachycardia, diaphoresis and confusion. The breath sounds may be normal. The x-ray may be insignificant and the arterial blood gases may show a moderate hypoxemia with normal or decreased P_aCO_2 and a normal pH. As the process worsens, the dyspnea and work of breathing will increase. The patient may become obtunded. Rales and rhonchi may be present. The x-ray will begin to show interstitial and alveolar fluid accumulation. The most decisive laboratory finding will be a severe hypoxemia that is not responsive to high concentrations of oxygen. Eventually, the patient develops respiratory failure at which point mechanical ventilation is initiated. (Note: Mechanical ventilation may be initiated much earlier in the process. The decision to begin ventilation depends on the patient's individual needs and the approach of the physician caring for the patient. The patient should be monitored carefully with frequent arterial blood gases and x-rays since the process can progress very rapidly.)

Management

The patient in respiratory failure should be intubated and placed on continuous mechanical ventilation. Electrolyte and acid-base abnormalities should be corrected as soon as possible. Serious metabolic acidemia resulting from lactic acid accumulation should be corrected promptly with sodium bicarbonate.

The most pressing problem in the management of ARDS is severe hypoxemia. The use of larger tidal volumes (15 ml/kg) in these patient has been found to improve the FRC, decrease the shunt fraction and improve oxygenation. The oxygen concentration should be established to maintain a P_aCO_2 around 50 mmHg. P_aCO_2's higher than 50 mmHg do not significantly add to the oxygen content since the oxyhemoglobin dissociation curve is flat at these levels of P_aCO_2. The goal in oxygen delivery is to maintain a P_aCO_2 of 50 mmHg with oxygen concentrations of 50% or less. When FIO_2's greater than 50% are required, PEEP should be initiated. Severe shunting may require PEEP levels up to 33 cmH_2O.

The patient's cardiovascular response should be monitored during the administration of PEEP. Ideally, the cardiac output should be monitored directly via a Swan-Ganz line. The cardiac output should be maintained using a combination of fluid therapy and pharmacological agents.

The administration of fluid therapy should be closely monitored since excess fluids can leak into the interstitial and alveolar lung spaces in ARDS. Monitoring should include determination of:

1. Fluid intake and output
2. Daily body weight
3. CVP determination
4. Pulmonary artery pressure
5. Capillary wedge pressure

(Determination of the pulmonary artery pressure and capillary wedge pressure requires a Swan-Ganz line.)

Some patients require sedation to control restlessness and anxiety. Sedative or narcotics can be used successfully. Remember, these drugs may affect the blood pressure.

Occasionally, some patients may require paralysis using curare or pancuronium bromide in order to maximize the benefit of the mechanical ventilator.

Management of the airway is usually not a problem since ARDS is not associated with increased pulmonary secretions. The patient should be routinely lavaged and suctioned to maintain a patent airway. Patients requiring extended mechanical ventilation should have a tracheostomy.

Complications

Many of the complications in the ARDS patient are common to any patient in an intensive care area receiving continuous mechanical ventilation (i.e., nosocomial infections, fluid overload, cardiac arrhythmias, etc.). ARDS patients on high levels of PEEP are at risk of developing pnuemothoraces. The other complication associated with high levels of PEEP is a reduction in the cardiac output.

Prognosis

In general, the more severe the initial insult, the poorer the prognosis in the ARDS patient. Patients with shunt fractions greater than 60% have a very poor prognosis. A better understanding of ARDS coupled with more sophisticated methods for managing the disease has significantly improved the prognosis for this patient group. Finally, the patient's prognosis is related to the ability of the medical, nursing and respiratory staff.

Blunt Chest Trauma

Etiology

Blunt chest trauma occurs as a result of the sudden, external application of a high speed object to the chest. Automobile accidents are responsible for the majority of blunt chest injuries. The sequelae of blunt chest trauma may include:

1. Hemothorax
2. Pneumothorax
3. Fractured ribs and/or sternum
4. Pulmonary contusions
5. Pulmonary lacerations
6. Airway ruptures
7. Laryngeal trauma
8. Subcutaneous emphysema
9. Diaphragmatic rupture
10. Esophageal rupture
11. Cardiac contusions, lacerations and ruptures

Clinical Manifestations

The patient suspected of having a blunt chest injury should be carefully examined and evaluated. The patient's entire chest and abdomen should be exposed and physically inspected for lacerations, bleeding and/or contusions. The respiratory pattern should be observed. Paradoxical movement of the chest indicates a flail chest. Paradoxical breathing is distinguished by an inward movement of the chest during inspiration. A flail chest usually occurs when the sternum is damaged or when more than three ribs have compound fractures. Such rib cage instability results in a reduction of the functional residual capacity and atelectasis. Because of the paradoxical motion of the chest during inspiration, the tidal volume can be reduced by more than 50%, resulting in a compensatory tachypnea. The presence of a hemothorax or pnuemothorax can further increase the atelectasis with the final result being severe hypoxemia, hypoventilation and acidosis, eventually leading to respiratory failure.

Management

Patients with simple rib fractures that do not result in a flail chest can usually be treated conservatively. These patients will have a reduced ventilatory capacity because of pain. This can be managed with narcotics or intercostal nerve-blocking agents. These patients can also be trained to deep breath effectively. With or without narcotics, the patient with minor rib fractures will need instruction, encouragement and assistance with their deep breathing and coughing. Permanent restriction of the chest wall by chest-strapping should be avoided as a means of reducing pain as it may cause atelectasis.

Patients having flail chest require immediate and aggressive medical support. The primary treatment of flail chest consists of endotracheal intubation and controlled mechanical ventilation with PEEP. If the patient will require mechanical ventilation for more than 10-14 days, a tracheotomy should be performed. No attempt should be made to wean the patient from the ventilator until the chest wall is stabilized and paradoxical breathing is absent or substantially diminished. A severe flail chest will require at least 5 to 10 days of mechanical ventilation in order to stabilize the affected area.

Pulmonary contusions are the most common sequelae of blunt chest injury. The contusions form within six hours after the initial injury and usually begin to resolve spontaneously within 2-3 days. Localized edema may be associated with the contusion. The presence of edema can cause right-to-left shunting and a reduction in the P_aO_2.

Pneumothoraces and hemothoraces are not uncommon in major chest injuries. A progressive tension pneumothorax can be particularly dangerous and requires immediate recognition and treatment. Tension pneumothoraces occur frequently as a consequence of positive pressure breathing in patients having chest injuries. Clinically, the patient with a pneumothorax or hemothorax will have dyspnea, tachycardia, tachypnea, and apprehension. Chest movement and breath sounds on the affected side will be diminished. Subcutaneous emphysema may also be present. Hemothoraces and pneumothoraces are definitively diagnosed with a chest x-ray.

Cardiovascular collapse often occurs in the patient suffering from a major chest injury. A decreased cardiac output and hypotension are usually the result of blood loss and hypoxemia. Internal hemorrhaging, either in the thorax or abdomen should be promptly located and corrected surgically. The patient may require up to 15-20 units of blood replacement during the first 24 hours of treatment.

Complications

Accompanying head and abdominal injuries increase the morbidity and mortality in patients with chest injuries. Patients with severe chest injuries should also be suspected of having possible cervical fractures and necessary precautions should be taken until proven otherwise.

Aspiration of stomach contents during the initial emergency treatment represents a serious complication resulting in pneumonitis. Lung contusions and open chest injuries often become infected and should be treated with appropriate antibiotic therapy. Shock, aspiration and long bone fracture are all contributing factors to ARDS.

Spontaneous tension pneumothoraces are often associated with flail chest patients requiring positive pressure breathing. The patient's breath sounds, clinical condition, arterial blood gases, and ventilator parameters should be monitored closely to identify the development of pneumothoraces.

Prognosis

The prognosis of the patient with chest trauma is related to the amount of chest trauma, the age of the patient, and other complications. Patients over 50 years old will require up to twice as much time on the ventilator as the 20 to 30 year old patient, all other variables being equal. This is due to the slow healing of the ribs and sternum in older patients.

Although the death rate associated with blunt chest injury is high, the actual cause of death is usually due to concurrent brain injury and/or abdominal complications.

Myasthenia Gravis

Etiology

Myasthenia gravis is a neuromuscular disease that is characterized by a failure of transmission of the impulse from nerve to muscle. The exact cause of myasthenia gravis is unknown. The disease is twice as common in woman as in men. The initial onset of the disease usually occurs around 25 years of age in women and 31 years in men. Myasthenia gravis is a chronic disease with periodic relapses and remissions. Emotional stress, respiratory tract infections, and other variables that generally add to the patient's level of stress may precipitate relapses.

Clinical Manifestations

Myasthenia gravis is characterized by a progressive muscular weakness and paralysis. It may affect any muscle in the body though the face, lips, tongue, and neck are most commonly affected. The respiratory muscles can become involved, resulting in respiratory failure. This happens in about 10% of patients with myasthenia gravis.

Management

Anticholinesterase drugs are the main component of treatment in myasthenia gravis. When the patient's vital capacity falls below 10-15 ml/kg of body weight, additional administration of anticholinesterase drugs such as neostigmine or edrophonium is contraindicated, since they may actually contribute further to the respiratory failure. At this point, the patient should be intubated and moved to an intensive care area. After he/she has been stabilized and arterial blood gases drawn to examine the acid-base status, edrophonium can be administered and the vital capacity serially measured to see if any improvement occurs. If the vital capacity does not improve, continuous mechanical ventilation may be indicated.

These patients have normal lungs and can be ventilated with low peak pressures and oxygenated well with low concentrations of oxygen.

Complications

For the intubated myasthenia gravis patient, meticulous care of the airway should be practiced to prevent avoidable respiratory infections. If the patient is on continuous mechanical ventilation, spontaneous respirations should be measured frequently to note changes. The length of ventilatory assistance can vary considerably, depending on the patient.

Management of the myasthenia gravis patient requiring surgery can be precarious. The use of curare should be avoided. Post-operatively, these patients are also sensitive to antibiotics with neuromuscular blocking action such as neomycin. Anticholinesterase drugs should be withheld for four or five days after surgery and restarted with caution. Post-surgical patients should be in a respiratory intensive care for close observation.

Prognosis

The prognosis is usually better in women than in men. Also, younger patients have a better prognosis. Patients requiring continuous mechanical ventilation have a poorer prognosis.

Guillain-Barré

Etiology

Guillian-Barré is a somewhat rare paralytic disease also known as idiopathic polyneuritis and acute post-infective polyneuritis. The exact etiology of Guillain-Barré is unknown. As the term "post-infective polyneuritis" implies, the disease usually follows a viral infection of the upper respiratory tract. The diagnosis of Guillain-Barré is confirmed by a lumbar puncture, which shows elevated levels of protein in the cerebral spinal fluid in most patients.

Clinical Manifestations

Guillain-Barré can usually be diagnosed by the patient's history - an upper respiratory tract infection followed by paralytic symptoms. The paralysis is usually ascending and symmetrical, though variations are possible. The patient will exhibit varying degrees of general muscle weakness, dysphagia, and speech difficulties. The paralysis quite often involves the respiratory muscles, leading to respiratory failure.

Management

Approximately half of the patients with Guillain-Barré develop respiratory failure and require continuous mechanical ventilation. Since recovery may take months, a tracheotomy should be performed early. Meticulous care of airway is mandatory to prevent pulmonary complications due to nosocomial infections. These patients should be ventilated using an assist/control mode. The ventilator should have low-pressure and low-volume alarms. These patients are quite easy to ventilate since the lungs are normal. They will not demonstrate any significant shunting and can be oxygenated with low oxygen concentrations. Lungs will have a normal compliance.

Since the patient may require assisted ventilation for several months, it is important that psychological well-being is stressed in the overall care plan. Needs to be assured that most people who have Guillain-Barré recover and go on to live normal lives. Perhaps the most frustrating aspect of care is communication. Since many of these patients may have almost total paralysis, the use of the arms and mouth will be diminished, making communication very difficult. As a result, many will require much time, attention, patience and understanding. When the patient begins to show signs of respiratory activity, spontaneous respiratory parameters should be taken to monitor his/her progress and to determine if ventilatory weaning can be initiated.
Since many will have received continuous mechanical ventilation for several weeks or months, the weaning process should not be hurried, since general muscle weakness will be present.

One of the most important aspects of management is Guillain-Barré is physiotherapy. Passive exercises should be continued throughout the paralytic phase of the disease.

Complications

The neurological process may have several other manifestations. Patients may exhibit problems with intermittent low blood pressure due to an interruption of the autonomic reflex arcs controlling circulation. This is particularly evident when the patient is turned often and a sudden drop in the blood pressure results. Fluctuation in the blood pressure can be prevented by turning the patient more gradually and administering fluids to maintain a central venous pressure of 5 cmH_2O.

Patients with Guillain-Barré are very often unable to sweat except over very small portions of their body. They are very susceptible to changes in the environmental temperature. Their body temperature and the ambient temperature should be monitored frequently.

Prognosis

Recovery from Guillain-Barré is usually complete and most patients are able to lead a normal life after recovery. A relapse of the disease can occur, although this is quite rare. The patient should be followed up with good physiotherapy until complete function of all joints is gained.

Pulmonary Edema

Etiology

Pulmonary edema is the abnormal and excessive movement of fluid from the vascular space into the interstitium, alveoli, and conducting airways of the lung. This movement and accumulation of fluid from the vascular space into the alveolar space and airways may be the result of:

1. An increased capillary hydrostatic pressure in the vascular space

2. A decreased osmotic pressure in the vascular space

or

3. A failure of the pulmonary lymphatic system

Typically, pulmonary edema is divided into two major categories: cardiogenic, and non-cardiogenic. Cardiogenic pulmonary edema, the most common form, results from an increased pulmonary vascular hydrostatic pressure, secondary to left ventricular failure. Acute and chronic myocardial disease are the most common cause of left heart failure. Left ventricular failure is accompanied by increased end diastolic volumes which results in an increased left atrial pressure, increased pulmonary venous pressures and eventually, increased alveolar capillary pressures. This increased hydrostatic pressure in the alveolar capillaries results in a movement of fluid into the interstitial space. The lymphatics in the lungs increase their function to remove excess fluid from the interstitial space, but eventually cannot keep up with the movement of fluid out of the vascular space. Ultimately, there is an overall movement of vascular fluid into the interstitium and alveoli.

Non-cardiogenic pulmonary edema may be associated with a variety of diseases. Functionally, non-cardiogenic pulmonary edema is caused by:

1. Decreased colloidal osmotic pressure

2. Alteration or damage to the alveolar capillary membrane

and/or

3. Decreased functioning of the pulmonary lymphatics

Blood contains large molecules, mainly albumin and globin, that attract water molecules. These molecules do not easily move across the capillary membrane and thus keep water within the vascular membrane via their attractive force, called osmotic pressure. If the quantity of these molecules, referred to as osmolarity, decreases, there will be a tendency for water to move out of the vascular space into the interstitial space. If the alveolar-capillary membrane becomes damaged, it is even possible for albumin and globin to enter the interstitial space, attracting water. Hypoxia seems to be a predisposing factor in alveolar-capillary membrane damage and leaking.

Any process that reduces the absorption of fluids in the lungs by the lymphatic system will increase pulmonary edema. Although the failure of the lymphatic system is not the usual cause of pulmonary edema, poor lymphatic function can contribute to other precipitating causes.

Some of the conditions that may produce non-cardiogenic pulmonary edema include:

1. Oxygen Toxicity
2. ARDS
3. Heroin Addiction
4. Cerebral Disorders
5. Rapid infusion of intravenous solutions
6. Near Drowning

By far, the most common cause of pulmonary edema is cardiac disease. An increase in pulmonary wedge pressure is a strong indication of left heart failure.

Clinical Manifestations

In the early stage of pulmonary edema, the primary symptoms are dyspnea and tachypnea, usually accompanied with paroxysmal nocturnal dyspnea. The blood pressure is usually normal or slightly elevated. Auscultation will reveal basilar rales and scattered expiratory wheezing. The chest x-ray may be unremarkable at this point. Arterial blood gases will reveal a normal acid-base status with an accompanying mild to moderate hypoxemia. Sputum production will be unremarkable at this stage. The patient will be restless and anxious.

As the edema progresses, the patient will become more dyspneic and tachycardic. Respirations will become labored, accompanied by grunting. Hypotension may occur.

The blood pressure may become difficult to obtain by auscultation. The patient will develop a persistent cough that produces a frothy, rust colored or pink sputum. Auscultation will reveal moist rales and gross wheezing throughout the lungs. The patient may become cyanotic, confused, and diaphoretic. The chest x-ray will reveal an enlarged heart shadow, engorgement of the pulmonary vessels, a diffuse haziness throughout the lung fields and a butterfly appearance. Pleural effusion, sometimes associated with pulmonary edema, may also be present on the x-ray. The arterial blood gases will reveal a normal and elevated P_aCO_2 with appropriate changes in the pH, and a moderate to severe hypoxemia. There may also be an accompanying metabolic acidosis due to hypoxia and lactic acid production. A significant elevation in the P_aCO_2 indicates severe cardiopulmonary failure.

Management

Acute pulmonary edema can have a gradual or sudden onset. Mild cases can be treated successfully with digitalis, diuretics, antiarrhythmia agents, sodium restriction, bed rest and monitoring. However, severe pulmonary edema represents a life-threatening situation requiring immediate medical intervention.

Initial treatment of severe pulmonary edema should include high concentrations of oxygen via mask. A rapid acting diuretic such as furosemide should be given IV to initiate prompt diuresis, thereby decreasing the plasma volume and ventricular filling pressure. Initial doses of digitalis should be given to increase the contractility of the heart. Morphine sulfate may be given IM to reduce anxiety. Morphine will also tend to reduce the respiratory rate, decrease the heart rate and lower the blood pressure. Rotating tourniquets on three extremities promotes pooling of blood in the extremities thereby reducing venous return. This decreased venous return results in a reduction of the left ventricular end-diastolic volume. Aerosolized alcohol (20-50% solution) may be given via IPPB to reduce the surface tension of the intra-alveolar secretions and improve oxygenation.

If the patient becomes lethargic or comatose and the blood gases show deterioration in the P_aCO_2 with an increased P_aCO_2 despite initial therapy, he/she should be intubated and placed on mechanical ventilation with PEEP. He should also be sedated to decrease his work of breathing and permit effective delivery of mechanical ventilation. The patient will continue to produce copious amounts of pink, frothy sputum requiring frequent suctioning. Arterial blood gases should be brought within a normal range. Severe metabolic acidosis should be treated with sodium bicarbonate. Inline administration of an aerosolized bronchodilator may have some effect on wheezing. Inline administration of aerosolized alcohol may help in reducing the edematous secretions. The insertion of arterial and Swan-Ganz lines is indicated in patients suffering from severe pulmonary edema.

<u>Complications</u>

If severe hypoxia persists for any extended period of time, damage to the brain, heart and kidneys may occur. Everything must be done to maximize cellular oxygenation. This includes adequate arterial oxygenation and maintenance of a good cardiac output. Even moderate hypoxia in the myocardium can result in life-threatening arrhythmias. Mild hypoxia in the kidneys can result in a reduction in the much-needed urine output.
Refractory heart failure may be associated with severe pulmonary edema. In some patients, poor left ventricular function persists despite therapy. If this occurs, other underlying causes may be present, such as mitral or aortic stenosis.

<u>Prognosis</u>

Even though the patient with acute pulmonary edema can become severely ill, total recovery is possible. Some with severe pulmonary edema on admission may dramatically improve within the first 24 hours. Others may require 3 to 4 days of therapy before significant improvement is seen. Most patients receiving mechanical ventilation respond well to weaning once their oxygenation status is improved. Those with underlying pulmonary disease or infections will require more intensive and prolonged ventilatory assistance and will have more difficulty during the weaning process. Those patients have a poorer prognosis and may have problems not only with correcting hypoxemia, but with correcting hypercapnia.

Post-Surgical Recovery

Thousands of minor elective surgeries are performed daily in this country without any subsequent respiratory complications. This section will focus on the patient receiving elective or emergency thoracic and cardiac surgery and the implications for respiratory management.

<u>Pre-Operative Evaluation</u>

Candidates for elective thoracic or cardiac surgery should be evaluated prior to surgery to determine their level of risk in terms of their respiratory system. Not only does the pre-surgical evaluation help in assessing the surgical risk, but the information obtained is useful in providing a baseline for managing the patient post-operatively.

The evaluation should include a medical history, physical examination, pulmonary function studies, and arterial blood gases. A history of smoking, pre-existing lung disease, excessive sputum production, dyspnea and exercise intolerance all increase the risk of mobidity and mortality in thoracic and cardiac surgery. Surgical candidates with pulmonary function studies showing moderate to significant reduction in flowrates and/or volumes, coupled with arterial blood gases that reveal hypoxemia and hypercapnia on room air, represent serious surgical risks. The decision for surgical intervention is determined by weighing the surgical risks against the benefits of surgery. Although the laboratory and clinical information provide guidelines for determining surgical risk, the decision for surgical intervention is a medical one that involves more than just clinical and laboratory assessment.

Management

Regardless of the level of risk, any patient having thoracic or cardiac surgery should be observed in an intensive care area post-operatively for at least 24 hours. Today, patients coming out of cardiac surgery and some thoracic surgery routinely have arterial Swan-Ganz lines in place. Most of these patients will be recovered in the intensive care area and will require mechanical ventilation for at least 4-12 hours. Patients with a history of pulmonary disease or respiratory infection prior to surgery may require several days of mechanical ventilation post-operatively.

Maintenance of a patent airway is a priority. This should include routine suctioning and lavaging of the endotracheal tube using sterile procedures and equipment. The endotracheal tube should be well secured with adhesive tape or a manufactured device. The position of the endotracheal tube should be confirmed with a chest x-ray upon admittance into the intensive care area. Patients requiring mechanical ventilation beyond 10-14 days should receive a tracheostomy.

Patients with a history of pulmonary disease may exhibit wheezing post-operatively in which case routine administration of aerosolized bronchodilators should be given. The inspired gas delivered by the ventilator should be humidified to prevent the accumulation of thick bronchial secretions. Any changes in the color or consistency of the patient's sputum should be noted. Patients with significant secretion retention may benefit from postural drainage and chest percussion. If a patient is expected to be on the ventilator for more than three days, secretions from the endotracheal tube should be collected and sent to the laboratory for routine culture and gram stain.

The patient's spontaneous ventilatory parameters should be frequently measured and recorded. Measurement of the spontaneous tidal volume, respiratory rate, minute ventilation, vital capacity and peak negative pressure should be included. Most post-operative patients will not have weaning problems; however, no patient should be removed from mechanical ventilation until the adequacy of their spontaneous ventilatory effort is determined and documented. In addition to exhibiting adequate spontaneous respiratory parameters, the patient should be alert and should be able to cough adequately. Patients with significant shunting and/or deadspace ventilation should not be weaned from mechanical ventilation. As a rough guide, the P_aO_2 should be 40 mmHg on 50% oxygen and the P_aCO_2 should be 40 mmHg with less than 10 lpm total minute ventilation during assist-control or SIMV ventilation.

Many cardiac patients today receive duramorphe in surgery to reduce pain post-operatively. These patients are able to cough more effectively and breathe deeper; however, because of the adverse effects of duramorphe on the respiratory center, spontaneous respirations in these patients should be monitored closely following extubation for the first 25 hours post-operatively. The patient's respiratory rate, pattern and chest expansion should be monitored and documented hourly. Any abnormalities should be noted and reported to the physician.

After extubation the patient should be encouraged to breathe deeply and cough. Incentive spirometry is a useful and effective adjunct in the cooperative patient with normal lungs. Other modalities such as aerosol therapy, bronchodilator therapy, and/or IPPB may be indicated in patients with underlying pulmonary disease or infections.

Complications

Atelectasis and pneumonia are frequent complications associated with thoracic and cardiac surgery. Frequent coughing and turning of the patient are essential. The patient's activity should be increased as soon as possible starting with sitting on the edge of the bed and advancing to walking in the hallway. Separation of surgical patients from medical patients also reduces the incidence of secondary infections.

Hypotension and cardiac arrhythmias can generally be treated successfully with pharmacological agents. Dopamine can be titrated intravenously to maintain a systolic pressure above 100 mmHg. Arrhythmias are aggravated by the presence of hypoxia, acidemia, hypokalemia and digitalis toxicity. Normalization of the patient's arterial blood gases and electrolytes will go a long way in preventing life-threatening arrhythmias. Premature ventricular contradictions (PVCs) represent the most frequently occurring arrhythmia and are treated with lidocaine. Sinus bradycardia can be controlled with atropine, isoproterenol or atrial pacing. Ventricular tachycardia is treated with lidocaine or electrical defibrillation.

Occasionally, post-operative cardiac patients will develop a cardiac tamponade. The clinical findings are those associated with shock including decreased cardiac output, decreased blood pressure, dyspnea and general deterioration of the patient's condition. Immediate surgical intervention is required.

One of the risks associated with any cardiac surgery involving extracorporeal oxygenation is the occurrence of CVAs during and after surgery. The incidence of CVAs correlates with the length of time on extracorporeal oxygenation. Age does not seem to be a predisposing factor.

Prognosis

The prognosis for the thoracic and cardiac surgery patient depends on the patient's general condition, age and the type of surgery. For example, otherwise healthy patients in the 45-60 year bracket recover well from uncomplicated coronary artery bypass surgery. Patients above 60 years of age with underlying lung disease or other medical problems have a higher incidence of morbidity and mortality. The prognosis for patients who suffer from severe CVAs is extremely poor.

INITIATION OF MECHANICAL VENTILATION

Criteria For Establishing Respiratory Failure

The specific disease entities that can be associated with the need for mechanical ventilation are numerous. It is perhaps better to think in terms of general categories that are associated with respiratory failure. The following grouping is offered:

1. Neuromuscular depression or dysfunction
2. Chest wall injury or dysfunction
3. Airway obstruction
4. Alveolar filling abnormalities
5. Increased V/Q abnormalities
6. Decreased V/Q abnormalities

Table 3 gives examples for each of these categories. These categories are not meant to be mutually exclusive. Indeed, in the clinical setting these categories often occur in combinations in the patient requiring mechanical ventilation.

Table 3

CLASSIFICATION OF DISEASES AND ABNORMALITIES PRECIPITATING RESPIRATORY FAILURE

<u>Neuromuscular Depression or Dysfunction</u>
Drug overdose
Myasthenia Gravis
Guillain-Barré
Cervical injuries
<u>Chest Wall Injury or Dysfunction</u>
Post-surgical
Crushed-chest injury
<u>Airway Obstruction</u>
Asthma
COPD
Foreign body aspiration
<u>Alveolar Filling Abnormalities</u>
Pulmonary Edema
Pneumonia
<u>Increased V/Q Abnormalities</u>
Decreased cardiac output
Shock
<u>Decreased V/Q Abnormalities</u>
ARDS

Pulmonary edema

The presence of respiratory failure is determined by evaluating the patient's clinical condition and laboratory results. The patient in frank or impending failure can present with disorientation, lethargy, or severe obtusion, depending on the particular disease and patient. The respiratory pattern can be quite variable and is not a good indicator of the patient's respiratory status. Certainly, if the patient is hyperpneic, hypoventilation should be suspected. Hyperpnea is not necessarily an indication that the patient's respiratory status is adequate. Indeed, a patient can have severe tachypnea and still hypoventilate. Breath sounds are helpful in determining the extent of respiratory involvement in a particular disease, but auscultation by itself is not definitive. A patient may have impending respiratory failure and have an unremarkable chest x-ray, as in the early stages of pulmonary edema and ARDS.

The clinical symptoms and some of the laboratory information do not always correlate well with underlying respiratory failure. Arterial blood gases establish the definitive diagnosis. A patient is said to be in respiratory failure if the PaO2 is less than 50 mmHg and the PaCO2 is greater than 50 mmHg, with an acute change in the pH. These criteria need to be adjusted for the COPD patient.

Establishment of an Airway

Establishment of an airway is the first step that should be taken in the patient with impending or frank respiratory failure. This involves the insertion of an endotracheal tube either orally or nasally. The endotracheal tube permits the application of positive pressure breathing and facilitates bronchopulmonary care. The diameter of the endotracheal tube should be as large as possible to reduce airway resistance during mechanical ventilation; however, it should not be so large that it must be forced through the larynx. The patient's respirations should be assisted with a bag-mask device and supplemental oxygen during the intubation procedure. After the endotracheal tube is in place, the chest should be osculated to determine proper position of the tube. After the tube position is verified via chest x-ray, it should be secured with adhesive tape. The cuff should be inflated with just enough volume to seal the airway: minimal occluding volume (MOV). The pressure in the cuff should not exceed 25 cmH2O.

Establishment of Initial Ventilator Parameters

Several factors must be taken into consideration in establishing the initial ventilator parameters. The blood gas values must be evaluated to determine the degree of ventilatory and oxygenation failure. The patient's clinical condition and disease process must be taken into consideration. If the patient is severely hypoxemic, the ventilator should be initially set on 100% oxygen and blood gases drawn in 20 minutes to determine the response. If the PaO2 comes back greater than 70 mmHg, the FIO2 can be reduced. It is better to over-oxygenate initially than to under-oxygenate. If the patient does not respond well to high concentrations of oxygen, 10 cmH2O of PEEP should be initiated.

The tidal volume is typically established according to the patient's body weight. As a rough guideline, the tidal volume should be 10 ml/kg body weight; however, larger tidal volumes may help prevent further atelectasis in the patient with ARDS. The patient with severe dead space ventilation may require higher tidal volumes and rates in order to maintain an adequate minute ventilation. This patient may require a minute ventilation exceeding 30 lpm to maintain a normal $PaCO_2$ In the patient with a severely elevated $PaCO_2$, it is important that it be brought back within normal limits gradually. A rapid decline can result in cardiac arrhythmias, cerebral ischemia, hypotension and over-correction, resulting in alkalosis.

The flow rate should be adjusted to establish an I:E ratio of at least 1:1.5. In the average ventilator patient, the flow rate demands do not exceed 40 lpm; however, in patients with high peak inspiratory demands, large tidal volumes and/or high respiratory frequencies will require increased inspiratory flow rates.

The tidal volume and sigh volume pressure limits should be set to prevent excessive pressure build-up in the lungs and to monitor sudden changes in the patient's compliance. The pressure limits should be set 10-20 cmH_2O above the peak cycling pressures during delivery of the tidal volume and sigh volumes respectively.

The ventilator should have as a minimum the following alarms:

1. Ventilator failure
2. Low-pressure and/or disconnect
3. Low tidal volume delivery

The alarms should be set to match the patient's individual ventilator parameters.

If the patient "fights" the ventilator initially, it may be necessary to sedate him in order to optimize the ventilatory response. Five mg of morphine sulfate is usually sufficient in most patients. Valium may also be used or alternated with morphine.

WEANING FROM MECHANICAL VENTILATION

Weaning from the ventilator should not be attempted until the patient's clinical condition is stabilized. Otherwise, the weaning process will prove futile and counter-productive. The patient's sensorium should be normal. Patients who are lethargic or comatose do not tolerate weaning procedures. Secretions and bronchospasm should be under control.

The cardiovascular system should be stabilized including pulse, arrhythmias, and blood pressure. The patient's baseline blood gases should be achieved using minute volumes less than 10 lpm and oxygen concentrations of 40% or less. Nutritional status should be adequate as undernourished patients are weak and do not tolerate the weaning procedure well.

Methods of Evaluation and Criteria

Ventilator discontinuance or weaning should begin as soon as possible without jeopardizing the patient's safety. This means there must be significant improvement in the underlying disease process that prompted mechanical ventilation in the first place.

Various guidelines have been developed to assess the ventilator patient's spontaneous ventilatory reserve. Although the criteria in these guidelines are useful in making clinical decisions, they should not be a substitute for clinical assessment of the patient's overall condition. The values for the following criteria are summarized in Table 4.

Table 4

CRITERIA FOR WEANING FROM MECHANICAL VENTILATION

Parameter	Criteria
PaO_2	> 70 mmHg on 40% oxygen
Acid-base status	normal
$P(A\text{-}a)O_2$	< 300 mmHg
Peak Inspiratory Pressure	> 20 cmH_2O
Spontaneous Minute Ventilation	< 10 lpm
Maximal Voluntary Ventilation	Double the resting minute ventilation
Spontaneous Respiratory Rate	< 30 bpm
Vital Capacity	10-15 ml/kg body weight
FEV_1	> 10 ml/kg body weight
Spontaneous Tidal Volume	> 200 ml
V_D/V_T Ratio	< 0.55
Shunt Fraction	< 15%

Arterial Blood Gas Values

Before the ventilator can be discontinued or weaning initiated, the arterial blood gases must be brought within normal limits. This means a normal acid-base status and a PaO2 greater than 70 mmHg on 40% oxygen. For the COPD patient, the blood gases should be returned to the values that existed prior to the acute exacerbation. This will mean a compensated respiratory acidosis with a PaO2 of 60-70 mmHg. COPD patients ventilated for a normal pH and PaCO2 will quickly develop an uncompensated respiratory acidosis when ventilator discontinuance or weaning is attempted.

Alveolar-Arterial Oxygen Gradient

The alveolar-arterial oxygen gradient P(A-a)O2, also provides information about the adequacy of the patient's oxygenation. The patient is placed on 100% oxygen while on the ventilator and arterial blood gases are drawn. A P(A-a)O2 less than 300 mmHg on 100% oxygen indicates adequate pulmonary oxygenation ability.

Peak Inspiratory Pressure

The peak inspiratory pressure (PIP) provides information about the patient's neuromuscular strength, and requires less cooperation than some of the other procedures. An aneroid manometer is attached to the proximal airway. The manometer should be attached to a "y" permitting the patient to breathe spontaneously. On command, the patient is asked to inspire maximally while the "y" is occluded, creating a negative pressure during the maneuver. A PIP greater than 20 cmH2O indicates adequate inspiratory muscular strength. The Bennett 7200 ventilator has a built in mode to determine the PIP without disconnecting the patient from the ventilator.

Minute Ventilation

The patient's spontaneous minute ventilation should be less than 10 lpm. The patient should be permitted to breathe spontaneously for a couple of minutes before the spontaneous minute ventilation is obtained. The Bear I, Bear II, Bear 5 and Bennett 7200 permit measurement of the spontaneous minute ventilation with the patient attached to the ventilator using the CPAP mode.

Maximal Voluntary Ventilation

This test is dependent on patient cooperation and effort; however, if the patient is able to double their resting minute ventilation, adequate respiratory reserve is present. The test should be performed for 15 seconds and the data extrapolated for one minute.

Respiratory Rate

During the spontaneous respiratory trial, the patient's respiratory rate should be less than 30 bpm. Respiratory rates greater than 30 bpm represent significant airway dysfunction and increased work of breathing.

Vital Capacity

The vital capacity is a relatively simple procedure and provides a good indication of the patient's neuromuscular strength. A vital capacity greater than 10-15 ml/kg body weight indicates adequate ventilatory reserve. A patient with this vital capacity should be able to sigh and cough effectively. This measurement can also be taken with the patient attached to the ventilator using the Bear I, Bear II, Bear 5, or Bennett 7200.

Forced Expiratory Volume in 1 Second

The Forced Expiratory Volume in 1 Second (FEV1) can be an important criteria in assessing the spontaneous ventilatory reserve in the COPD patient. It is possible for these patients to have a near normal vital capacity and still have severe airway obstruction, which limits their ability to breathe spontaneously. These patients should have a FEV1 of at least 10 ml/kg body weight in order to cough effectively and prevent atelectasis.

Tidal Volume

In general, the spontaneous tidal volume is not a reliable indicator of the patient's ventilatory reserve. Certainly, a patient with a spontaneous tidal volume less than 100 ml will not tolerate ventilator discontinuance or weaning well. Patients with tidal volumes greater than 200 ml normally have adequate ventilatory reserve.

VD/VT

The VD/VT should be less than 0.55 if ventilator discontinuance or weaning is contemplated. Values greater than this represent significant dead space ventilation and may result in hypercapnia during spontaneous breathing.

Shunt Fraction

The shunt fraction should be less than 15%. Values greater than this represent significant venous admixture and unresolved pulmonary pathology. At shunt fractions above 15% the patient may not be able to oxygenate adequately during spontaneous ventilation.

Other Factors

There are several other factors that should be considered prior to discontinuing mechanical ventilation or initiating weaning. These factors are briefly discussed below.

Psychological Preparation

The patient should be instructed about the weaning procedure or ventilator discontinuance. During spontaneous breathing, the patient must be encouraged and relieved of apprehension. He/she must have confidence in the practitioner in charge of the ventilator management.

Cardiovascular Status

The patient's cardiovascular status must be stabilized. The hemoglobin should be above 10 g/100 ml to assure adequate oxygenation. The cardiac output should be maximized and arrhythmias should be under control. Significant tachycardia is a contraindication to ventilator discontinuance or weaning.

Nutrition

Long-term ventilator patients should be on a program that includes proper nutrition and passive exercise to increase muscle strength and energy. These should be initiated early in the management of the patient.

Fatigue

The patient should be given opportunities to rest. If spontaneous breathing trials are attempted, he should be allowed to rest on the ventilator at night. This will tend to shorten the total length of time on the ventilator.

Fluid Balance

The intake and output (I&O) must be carefully monitored in the ventilator patient. It is known that positive pressure breathing causes an increase in antidiuretic hormone (ADH) resulting in a decreased urine output. In addition to strict I & O measurements, the long-term ventilator patient should be weighed daily to monitor weight gain due to excess fluid.

METHODS OF WEANING

There are several approaches that can be used to discontinue ventilatory support. Most ventilator patients can be removed from mechanical ventilation without a weaning process. The various approaches are discussed below. It is quite possible that a combination of modalities might be beneficial in some patients.

Ventilator Discontinuance

Most patients who have received short term mechanical ventilation do not need a gradual process of weaning. In these patients it is better to think in terms of ventilator discontinuance rather than weaning. Certainly, post-operative patients who are on the ventilator for less than 24 hours do not need to be weaned. Usually they will begin to demonstrate adequate spontaneous respiratory parameters as they become more alert. As a general rule, the ventilator should be discontinued as soon as it is feasible without sacrificing the patient's safety. If the spontaneous respiratory parameters are adequate and the other pertinent clinical parameters do not present any contraindication, the patient should be removed from the ventilator and placed on a Briggs "T" piece at the same FIO2 or at an FIO2 10% higher than the ventilator setting. He/she should be monitored closely for signs of respiratory and cardiovascular insufficiency. If he tolerates 20 minutes off the ventilator, an arterial blood gas should be drawn to assess his ventilation and oxygenation. If the blood gases are satisfactory the patient need not remain intubated unless it is felt that he should be evaluated on the Briggs "T" piece for a longer period of time. After the patient demonstrates an adequate spontaneous respiratory reserve, he can be extubated and placed on an aerosol device. An additional arterial blood gas should be obtained after approximately one hour on the aerosol to reconfirm the adequacy of the patient's spontaneous ventilatory effort. Other patients may not respond as well to the first effort to discontinue mechanical ventilation. If this occurs, the patient should remain on the ventilator until the spontaneous respiratory parameters improve.

Spontaneous Breathing Trials

This method is essentially just an extension of ventilator discontinuance. A regimen is established in which the patient is periodically placed on a Briggs "T" piece for increasing periods of time. As a patient's spontaneous ventilatory capacity improves, the frequency and length of spontaneous breathing is increased until the patient can tolerate an hour on the Briggs "T". Usually, once a patient can tolerate 1-2 hours on the Briggs "T" piece, they progress very rapidly and are off the ventilator in a few days. During the spontaneous breathing trials, the patient should be monitored closely for signs of respiratory distress and cardiovascular complications such as tachycardia, hypotension and arrhythmias. An expired CO_2 monitor is also helpful for preventing dangerous increases in the $PaCO_2$.

SIMV

SIMV can be a useful modality for weaning the more difficult patient from mechanical ventilation. It has the advantage of providing a pre-set minute ventilation, which is synchronized with the patient's spontaneous ventilatory efforts. As the patient's ventilatory capacity improves, indicated by the objective ventilatory parameters, the SIMV rate is gradually reduced until the total exhaled minute volume is produced largely by the patient's spontaneous respiratory activity. At this point, the patient can be placed on spontaneous ventilation through the ventilator and arterial blood gases obtained to verify the adequacy of the patient's ventilatory effort. If the patient is able to maintain stable arterial blood gases and adequate ventilatory effort without showing signs of increased work of breathing, the patient can be extubated and placed on an aerosol device.

SIMV is a useful modality; however, not every patient requires SIMV, nor is SIMV a panacea for the patient with a minimal spontaneous respiratory capacity.

Pressure Support Ventilation

Pressure support ventilation (PSV) is a relatively new modality that appears to have application in weaning from mechanical ventilation. PSV can be used by itself in a spontaneous mode or used in conjunction with SIMV. The use of PSV allows the patient with an inadequate spontaneous ventilatory capacity to tolerate SIMV and spontaneous breathing modes better, both subjectively and in terms of objective criteria.

AMV (Bear 5)

Augmented minute ventilation (AMV) is another new ventilator modality currently available on the Bear 5 ventilator. It is very similar to SIMV except that a minimal total exhaled minute volume is guaranteed. This helps to eliminate precipitous increases in the PaCO2 that can occur in SIMV and spontaneous breathing modes when the patient's spontaneous minute ventilation suddenly decreases. AMV appears to provide another modality for weaning the more difficult ventilator patients.

NUTRITIONAL SUPPORT

Extended stays in the hospital are often associated with malnutrition. Critically ill patients maintained on sophisticated life-support systems for long periods of time exhaust their nutritional reserves. Patients who become malnourished have a prolonged hospital stay and a higher mortality. Malnourished patients do not tolerate illness well, tend to experience delayed wound healing, and are more susceptible to infections. Very often malnutrition is an iatrogenic disorder and is due to an emphasis on the patient's primary problem and neglect of nutritional concerns.

Factors Contributing to Malnutrition

Malnutrition can be due to one or more of the five factors listed below:

1. Inadequate dietary intake, particularly caloric and protein
2. Inadequate nutritional absorption
3. Inadequate utilization of nutrition in its cell
4. Increased losses
5. Increased requirements

Of these five factors, inadequate dietary intake and increased requirements are responsible for most of malnutrition seen in ventilator patients. The dependence on IV glucose as the primary means of nutritional support can result in inadequate dietary intake. Tissue hypoxia, fever, surgery, trauma, and infection all increase the dietary requirements of the ventilator patient. For example, fever can increase the caloric intake required to maintain adequate nutrition by as much as 50%. It is not unusual for the febrile patient to have an actual caloric requirement of 5000-7000 kcal/day in the intensive care area.

Methods of Nutritional Support

There are two main modes for providing nutritional support: internal and parenteral. The entail mode includes oral and nasogastric tube feeding. The parenteral mode includes peripheral venous and central venous alimentation. The specific mode(s) used depends on the patient's clinical situation and nutritional needs.

In general the most physiological route should be used to administer nutritional support if the patient can tolerate it. This means oral intake is preferred. If the patient has a decreased appetite or inability to take foods orally (e.g., an endotracheal tube in place), then nasogastric feeding is the next desirable route. The nasogastric route is effective in many patients, particularly when short-term nutritional support is required.

Evaluation of Nutritional Support

Body weight is the simplest method of evaluating the nutritional status of the patient. Weight loss that is not secondary to fluid loss usually indicates inadequate nutritional intake. When the caloric intake does not meet the nutritional demands of the patient, the body begins to convert proteins residing in the muscle tissue into carbohydrates, resulting in muscle loss and weakness. The conversion of fats and proteins into carbohydrates occurs in patient who are maintained on IV solutions of glucose for long periods of time. It is often assumed that the obese patient requires less nutritional support. This misconception results from the well-fed appearance of the obese patient; however, it is possible for the obese patient to suffer protein-caloric malnutrition if improperly managed.

Nutritional Support in the Ventilator Patient

In the past the nutritional needs of the long-term ventilator patient were often overlooked. Studies have shown that inadequate nutrition increases the length of time on the ventilator and increases mortality. Patients who are well nourished tolerate the course of ventilator therapy better, improve more quickly, and tolerate weaning better due to increased muscular strength.

Muscle weakness and atrophy can occur in the long-term ventilator patient due to two factors:

1. The muscles can atrophy due to disuse.

The muscles may be cannibalized to meet the energy requirements of the body.

Muscle weakness and atrophy in the ventilator patient can be reduced by a treatment plan that includes adequate nutritional support and physical therapy.

Studies have shown that excessive glucose administration in the malnourished ventilator patient increases the respiratory quotient (VCO_2/VO_2), resulting in an increased CO_2 production and an increased demand on the respiratory system. This can become a particularly significant factor during the weaning process.

The weight should be closely monitored in the long-term ventilator patient. Weight loss not due to fluid loss is due to inadequate nutrition. Serial determinations of the circumference of the arm muscle can also be helpful in monitoring the adequacy of nutritional support. A decrease in the circumference of the arm muscle by more than 1.5 cm indicates the possibility of inadequate nutrition; however, indirect calorimetry using a metabolic cart is the most reliable and accurate technique for estimating the caloric requirements of the ventilator patient.

STUDY OUTLINE

I. Introduction

A. The intensive-care setting
1. An acute care environment for severely ill patients
2. Units may be specialized in larger hospitals
3. Maximized levels of care
a. The patient is close to staff
b. ICU staff are trained and certified to administer the latest critical care procedures and medications
c. ICU incorporates the latest monitoring and therapeutic equipment

B. The health-care team
1. Communication between people and departments to assure a comprehensive and integrated treatment plan

II. Ventilator Procedures

A. SIMV
1. A mechanical ventilator mode that allows the patient to breathe spontaneously and/or receive a volume from the ventilator at predetermined intervals
a. Spontaneous and mandatory breaths should be identical in oxygen concentration, humidity and temperature
2. Modern machines
a. Most provide SIMV rather than IMV
3. In weaning, SIMV is better than IMV both physiologically and psychologically
a. It is important to keep records distinguishing the ventilator rate, tidal volume and minute ventilation from the patient's spontaneous rate, tidal volume and minute ventilation
b. As SIMV rate is reduced, total minute ventilation should remain about the same

B. PEEP
1. A ventilator modality whereby a constant positive pressure is applied to the lungs during the expiratory phase
2. It improves oxygenation in severely hypoxic patients
a. It should be considered when PaCO2 remains below 50 mmHg on concentrations of oxygen greater than 50%
b. during IPPV
3. Application of PEEP
a. To patient with acute restrictive disease and reduced FRC
1) Alveolar collapse leads to physiologic shunting and hypoxemia
2) Decreased compliance and increased work of breathing
b. PEEP with improved FRC, and reduce the work of breathing by improving lung compliance

4. Disadvantages
    a. A reduction in cardiac output due to increased intrathoracic pressures, leading to a decrease in tissue oxygenation
5. Evaluating the effectiveness of PEEP
    a. Swan-Ganz line to determine "optimal PEEP" and/or arterial blood gases, pulse, blood pressure, and clinical assessment to determine "best PEEP"
    b. Weaning involves a reduction of FIO2 followed by reducing PEEP

C. CPAP
1. Similar to PEEP, but used in conjunction with spontaneous breathing
2. For patients who are severely hypoxemic despite high O2 concentrations, but capable of maintaining adequate minute ventilation and PaCO2
3. Is administered via a cuffed endotracheal or tracheostomy tube
4. Weaning is identical to PEEP procedures

D. Pressure support ventilation
1. Augments spontaneous inspiratory effort using an operator adjustable positive pressure; applied only during the inspiratory phase
    a. The patient determines gas flow and pressure breath by breath demands
2. Applications
    a. Low pressure during PSV to overcome the resistance to gas flow through the endotracheal or tracheostomy tube
        1) Used with SIMV or spontaneous breathing
    b. Uses pressures higher than needed to overcome the resistance to flow through the endotracheal or tracheostomy tube
        1) Used with SIMV or spontaneous breathing

E. Augmented minute ventilation
1. Establishes a back-up minute ventilation
2. Application is in weaning difficult patients from mechanical ventilation
    a. Helps prevent dangerous fluctuations in PaCO2 that can occur during SIMV and spontaneous breathing

III. Patient Evaluation

A. Bedside assessment procedures
1. Basic techniques
    a. Observation
    b. Palpation
    c. Percussion
    c. Auscultation

B. Neurological assessment
1. Moderate hypoxemia produces dyspnea and often anxiety

2. Severe hypoxemia causes disorientation , lethargy, and coma
    a. Can develop slowly or suddenly

C. Pulmonary assessment
1. Respiratory pattern
    a. Rate and depth of breathing
    b. I:E ratio (should be 1:2)
    c. Presence or absence of accessory muscle activity
    d. Bilateral chest expansion
    e. In chest trauma, look for "flailing"
2. Chest auscultation
    a. To identify intensity, quality and pitch of breath sounds
    b. Sound quality may be vesicular, bronchial, or adventitious
        1) Vesicular is normal, with inspiration louder, higher-pitched, and longer than expiration
        2) Bronchial is loud, harsh, and tubular, with expiratory longer and louder than inspiratory with a pause in between
        3) Adventitious includes rales, rhonchi, wheezing, and pleural rubs
    c. Distribution and intensity of breath sounds
        1) Vertically, sounds are less intense in the bases
        2) Horizontally, right and left lungs are equal
3. Sputum
    a. Qualities of volume, consistency, purulence, color, odor, presence of blood
    b. Characteristics
        1) COPD patient has large amounts
            a) So do abscesses, cystic fibrosis, and lung cancer
        2) Asthmatic patient has an increase in clear, mucoid secretions
        3) Infection by pathogenic bacteria produces purulent secretions
            a) Seen in pneumonia, acute bronchitis, and aspiration of pneumonia
        4) Lung abscess, bronchiectasis, cystic fibrosis, and aspiration pneumonia produces a fetid sputum
        5) Tuberculosis, carcinoma of lung, and bronchiectasis produce bright red blood in sputum
        6) Some pneumonias produce rusty or darker red blood in sputum
        7) Blood may appear in sputum from other areas of the body as well

4. Dyspnea
    a. A subjective perception that cannot be quantified
5. Other observations
    a. Cyanosis
        1) May not indicate true oxygen status

D. Cardiovascular assessment
1. Arterial line
    a. Attached to a pressure transducer; a wave is produced on an oscilloscope
    b. Gives systolic and diastolic pressures, and mean arterial pressure
    c. Allows for frequent blood gas analysis and withdrawal of blood for other lab tests
2. CVP line
    a. Assesses the relationship between the heart's pumping action and circulatory blood volume
    b. Changes in values
        1) A serial change is more significant than any one value
        2) Declining CVP may indicate hypovolemia or an improvement in the patient's cardiac output (if condition is otherwise improved)
        3) A high CVP may indicate fluid overload or cardiac failure
    c. CVP data is useful only if right and left heart are functioning equally
3. Pulmonary artery catheter
    a. The Swan-Ganz line is a means of measuring pulmonary artery pressure, wedge pressure, and cardiac output

IV. Bedside Monitoring Devices

A. Oximeters
1. Allows continuous, non-invasive monitoring of oxygen saturation
2. Polarographic units use a Clark electrode to sense the PaO2 on the skin's surface
3. Spectrophotometric units use light absorption to approximate the amount of oxyhemoglobin in the blood
4. Indicated in patients with decreased or fluctuating oxygenation, and as a way to reduce the number of arterial blood gases needed

B. CO2 monitors
1. Can be measured by infrared irradiation, thermal conductivity, the Severinghaus electrode, or mass spectrometry
    a. Most bedside equipment uses infrared irradiation on the gas sample
2. Indicated in the unstable ventilator patient with hypercapnia
3. Also used to evaluate changes in the ventilator minute ventilation, during weaning, and to reduce the need for arterial blood gases
4. The monitor's sampling line should be as close as possible to the

proximal airway

V. Laboratory Assessment Procedures

A. Arterial blood gas analysis
1. PaO2 is a direct measurement of the amount of dissolved oxygen in the arterial blood
    a. Combined with CaO2 and cardiac output, it gives an evaluation of tissue oxygenation
2. PaCO2 assesses alveolar ventilation
    a. An increase in PaCO2 shows a decrease in the alveolar ventilation
3. Distinguishing respiratory compensation from metabolic compensation
    a. The patient's clinical history is necessary

B. Chest x-rays
1. Bony structures
    a. Examine the rib cage for abnormalities
        1) Ribs should be spaced equally
        2) Compression on one side could indicate atelectasis on that side
        3) Rib evaluation could be due to airway destruction
        4) Note fractures or breaks
2. Pleural space
    a. Abnormalities include the presence of air or fluid, masses, or blunting of costophrenic angle due to fluid accumulation
3. Heart
    a. Size can indicate congestive heart failure
    b. Normal width is no more than half the distance across the lungs at the level of the diaphragms
4. Diaphragm
    a. Taken at full inspiration, they should be lower and flattened, (right side slightly higher than the left)
    b. Abnormal evaluation may indicate atelectasis
    c. Abnormal flattening may be from acute or chronic airway obstruction
5. Lung fields
    a. Examine for overall translucency
    b. Increased translucency in an area reflects pneumothorax, blebs, asthma, and COPD
    c. Decreased translucency shows that fluid or atelectasis is present
6. Airways
    a. Tracheal position should be midline
    b. Deviation may be due to poor positioning (compare to clavicles)
    c. Deviation also caused by atelectasis, pneumothorax, collapsed lung segments or lobes

VI. Situations Requiring Ventilator Assistance

A. COPD

1. Etiology
    a. Typical diseases are chronic bronchitis, emphysema, bronchiectasis, asthma
    b. Patient history includes chronic cough, sputum production, wheezing, and rhonchi
        1) Chest is hyperextended
        2) Accessory muscles are used
        3) Prolonged expiration
        4) Tachycardia
        5) Possible cyanosis
        6) X-ray shows hyperexpansion, flattened diaphragms, elevated ribs, and general translucency
        7) Arterial blood gases show compensated respiratory acidosis and hypoxemia on room air
    c. Respiratory failure may occur secondary to infection, bronchospasm, or heart failure
        1) At first, patient may be anxious and confused; in later stages lethargic and comatose
3. Management
    a. Intubate and place on continuous mechanical ventilation
        1) Gradually increase patient's CO2 to improve the pH
    b. If metabolic acidosis is present, correct with sodium bicarbonate
    c. Be careful not to over-ventilate
        1) Is helpful to know patient history of baseline values and pulmonary function studies
    d. Because several days of ventilation are usually required, nasal intubation is preferred
        1) Consider tracheotomy if ventilation will continue over 10-14 days
    e. Wheezing may be severe
        1) Digitalis, diuretics, antibiotics, and bronchodilation therapy may help
        2) It is important to identify the cause of the wheezing before attempting to treat
    f. Weaning should not be attempted until the respiratory and cardiovascular systems are stabilized
4. Complications
    a. Nosocomial infections
    b. Pneumothorax (emphysema)
    c. Emotional reactions
    d. Ventilator dependence
5. Prognosis
    a. Quite variable, depending on age, severity, and other medical problems
    b. An aggressive and continuing rehabilitation program

should include:

1) Instruction about the disease
2) Pharmacological therapy
3) Good hygiene
4) Breathing instruction
5) Exercise
6) Improved Nutrition
7) Periodic evaluation and adjustment of treatment

c. Chronic bronchitis

1) A chronic cough with excessive sputum
2) Persists 3 months or more for two or more consecutive years
3) Hypertrophy of mucus glands and goblet cells, and decreased ciliary activity
4) High correlation with cigarette smoking

d. Emphysema

1) Nonreversible destruction of pulmonary septal walls and connective tissue beyond the terminal bronchioles
2) Loss of elastic recoil; alveolar hyperdistension
3) Occurs often with chronic bronchitis

e. Bronchiectasis

1) Abnormal dilation and distortion of bronchi and/ or bronchioles
2) Associated with increased cough and purulent sputum

f. Asthma

1) Intermittent and reversible, with an unknown etiology
2) Precipitated by infections, emotional stress, allergens, cold weather, exercise
3) Symptoms are wheezing and dyspnea
4) Managed by bronchodilators and steroids

B. Asthma

1. Etiology
    a. Unknown, obstruction occurs in both the large and small airways
    b. Status asthmaticus is an acute exacerbation that is non-responsive to bronchodilation therapy, and is life-threatening
2. Clinical Manifestations
    a. Wheezing associated with a lengthening expiration
    b. Productive coughs with thick, clear, mucoid sputum
    c. Moderate distress, dyspnea
    d. Chest is over-distended and hyperresonant
    e. As the asthma progresses, patient begins to retain CO_2 and will be hypoxic on supplemental oxygen, fatigued and obtunded, with cyanosis (respiratory failure may be impending)

3. Management
    a. A regimen of aerosolized or oral bronchodilators, decongestants, steroids, and allergy-blocking agents
    b. Reduced environmental irritants and emotional stress
    c. Patient with a $PaCO_2$ greater than 55 mmHg and PaO_2 less than 60 mmHg on 50% oxygen, who does not respond to bronchodilator therapy, should be intubated and placed on continuous mechanical ventilation
        1) Morphine sedation to reduce anxiety and improve efficiency of ventilation
        2) Should require only 3-4 days of mechanical ventilation
4. Complications
    a. Secondary viral or bacterial infections
    b. Spontaneous pneumothorax from air-trappings and hyperinflation of alveoli
5. Prognosis
    a. Very good, particularly in children

C. ARDS
1. Etiology
    a. Factors include oxygen toxicity, near drowning, severe pneumonitis, trauma, post-cardiopulmonary bypass, and shock
    b. Onset is within 12-48 hours of injury
2. Clinical manifestations
    a. Increased permeability of alveolar-capillary membrane (fluid leaks into alveolar space)
    b. Increased workload on right heart
    c. Reduced surfactant production, causing alveolar collapse, shunting, and decreased compliance
    d. Patient initially shows mild dyspnea, increased work of breathing, tachycardia, diaphoresis, and confusion
3. Management
    a. Intubate and place on continuous mechanical ventilation
    b. Correct electrolyte and acid-base abnormalities
    c. Use of larger tidal volumes improves oxygenation
    d. When FIO_2 over 50% is required, initiate PEEP
        1) Monitor cardiovascular response
        2) Monitor fluid therapy
    e. Sedation may be needed to control restlessness and anxiety
    f. Airway management is usually not a problem, since secretions are not increased
4. Complications
    a. Nosocomial infections
    b. Fluid overload
    c. Cardiac arrhythmias
    d. Pneumothorax
    e. Reduced cardiac output
5. Prognosis
    a. The more severe the initial insult, the poorer the prognosis

(especially if shunt fraction is over 60%)

D. Blunt Chest Trauma

1. Etiology
    a. Automobile injuries are responsible for the majority of blunt chest injuries
    b. Sequelae include:
        1) Hemothorax
        2) Pneumothorax
        3) Fractured ribs and/or sternum
        4) Pulmonary contusions
        5) Pulmonary lacerations
        6) Airway ruptures
        7) Laryngeal trauma
        8) Subcutaneous emphysema
        9) Diaphragmatic rupture
        10) Esophageal rupture
        11) Cardiac contusions, lacerations, and ruptures
2. Clinical manifestations
    a. Requires careful examination and evaluation
        1) Paradoxical chest movement indicates flail chest
        2) Paradoxical breathing is an inward movement of the chest during inspiration
        3) These injuries reduce the FRC and cause atelectasis
    b. A hemothorax or pneumothorax can further increase atelectasis, leading to severe hypoxemia, hypoventilation, and acidosis, leading to respiratory failure
3. Management
    a. Simple rib fractures not resulting in flail chest
        1) Treat conservatively
        2) Manage pain with narcotics or intercostal nerve blocking agents
        3) Train in effective deep breathing and coughing
        4) Chest strapping may promote atelectasis
    b. Flail chests
        1) Require immediate and aggressive medical support
        2) Endotracheal intubation and controlled mechanical ventilation with PEEP
        3) Do not wean until chest wall is stabilized and paradoxical breathing is absent or substantially diminished
    c. Pulmonary contusions
        1) From 6-8 hours after the injury
        2) Localized edema can cause right-to-left shunting and reduce PaO_2
    d. Pneumothorax
        1) Tension pneumothorax occurs from positive pressure breathing in chest injury patients
        2) Clinical signs are dyspnea, tachycardia, tachypnea, and apprehension

3) Chest movement and breath sounds on the affected side may be diminished
4) Subcutaneous emphysema may be present

e. Cardiovascular collapse
1) Usually the result of blood loss and hypoxemia
2) Internal hemorrhaging must be surgically corrected, 15-20 units of blood may be required during the first 24 hours

4. Complications
a. Head and abdominal injuries
b. Cervical fractures
c. Aspiration of stomach contents
d. Infections

5. Prognosis
a. Is related to amount of injury, age, and other complications
b. The death rate is high, but the actual cause of death is usually due to concurrent brain injury and/or abdominal complications

E. Myasthenia Gravis

1. Etiology
a. A neuromuscular disease in which there is failure of the transmission of the impulse from nerve to muscle
b. Twice as common in women; onset is age 25 (in mean, age 31)
c. Chronic, with relapses and remissions

2. Clinical manifestations
a. Progressive muscular weakness and paralysis
b. Face, lips, tongue, and neck are most commonly affected
c. Respiratory muscles become involved in approximately 10% of patients

3. Management
a. Anticholinesterase drugs
1) Become contraindicated if vital capacity falls below 10-15 ml/kg of body weight
b. Then, intubate and move to intensive care
c. If vital capacity cannot be improved, continuous mechanical ventilation may be needed

4. Complications
a. Respiratory infections
b. Post-operative patients are sensitive to curare, antibiotics with neuromuscular blocking action, and anticholinesterase drugs

5. Prognosis
a. Usually better in women than men, better in younger patients
b. Those requiring continuous mechanical ventilation have a poorer prognosis

F. Guillain-Barr

1. Etiology
a. A somewhat rare paralytic disease, also called idiopathic polyneuritis or acute post-infective polyneuritis
b. Exact cause is unknown, but it follows a viral infection of

the upper respiratory tract

c. Confirmed by a lumbar puncture showing elevated levels of protein in the CSF

2. Management
    a. Approximately 50% will require continuous mechanical ventilation
        1) Recovery may take months
    b. Patients are easy to ventilate; lungs are normal
        1) Use low-pressure, low-volume alarms
    c. Stress their psychological well-being
        1) Most will recover
    d. With almost total paralysis, communication is difficult and frustrating
    e. Physiotherapy (passive exercises) should be continued throughout the paralytic phase of the disease
3. Complications
    a. Intermittent low blood pressure
    b. May be unable to sweat over much of the body
4. Prognosis
    a. Recovery is usually complete; relapses are rare

G. Pulmonary edema
1. Etiology
    a. May be the result of:
        1) Increased capillary hydrostatic pressure in the vascular space
        2) Decreased oncotic pressure in the vascular space
        3) Failure of lung's lymphatic system
    b. Cardiogenic pulmonary edema
        1) Increased pulmonary vascular hydrostatic pressure, secondary to left ventricle failure
    c. Non-cardiogenic pulmonary edema
        1) Decreased colloidal osmotic pressure
        2) Alteration or damage to the alveolar capillary membrane
        3) Decreased functioning of the lymphatics in the lung
        4) Diseases causing this include:
            a) Oxygen toxicity
            b) ARDS
            c) Heroin addiction
            d) Cerebral disorders
            e) Rapid intravenous infusion
            f) Near drowning
2. Clinical manifestations
    a. In early stages, dyspnea and tachypnea, normal or slightly elevated blood pressure, basilar rales, and scattered expiratory wheezing
    b. Later, increased dyspnea and tachycardia, labored respirations, hypotension, persistent cough, with frothy rust-colored and pink sputum, moist rales, and gross wheezing. Patient is cyanotic, confused and diaphoretic

c. Chest x-ray will show enlarged heart shadow, engorged pulmonary vessels, and diffuse haziness in lung fields, butterfly appearance, and perhaps pleural effusion
d. Arterial blood gases show normal or elevated $PaCO_2$, with changes in pH and hypoxemia, perhaps a metabolic acidosis

3. Management
   a. Treat mild cases with digitalis, diuretics, antiarrhythmia agents, sodium restriction, bed rest, and monitoring
   b. Treat severe edema with oxygen via mask, diuretics via IV, digitalis, morphine, rotating tourniquets, and aerosolized alcohol via IPPB
   c. Lethargic or comatose patient with deteriorating PaO_2 and increased $PaCO_2$ requires intubation and mechanical ventilation with PEEP, sedated and given frequent suctioning
      1) Sodium bicarbonate for severe metabolic acidosis
      2) Inline administration of aerosolized bronchodilator for wheezing, alcohol for reducing secretions
      3) Arterial and Swan-Ganz lines are indicated
4. Complications
   a. Damage to brain, heart, and kidneys
5. Prognosis
   a. Total recovery is possible
   b. Patients with underlying pulmonary disease or infections require more intensive and prolonged ventilatory assistance, and have a more difficult weaning, poorer prognosis

H. Post-surgical recovery

1. Pre-operative evaluation
   a. To determine the level of risk to the respiratory system and to establish a baseline
   b. Include medical history, physical exam, pulmonary function, and arterial blood gases
2. Management
   a. Requires 24 hours post-operative observation
   b. Maintain a patent airway
      1) Routine suctioning and lavaging of endotracheal tube
      2) Confirm position of tube with x-ray
   c. Those with history of pulmonary disease may require aerosolized bronchodilators, postural drainage, and chest percussion
   d. Frequent measurement and recording of spontaneous ventilatory parameters
      1) Cardiac patients who received duramorphe require close observation because of its effects on the respiratory center
   e. Incentive spirometry after extubation is good for cooperative patient with normal lungs

3. Complications
    a. Atelectasis
    b. Pneumonia
    c. Secondary infections
    d. Hypotension and cardiac arrhythmias
    e. Cardiac tamponade
    f. CVA
        1) Incidence is correlated with length of time on extracorporeal oxygenation
4. Prognosis
    a. Depends on general condition, age, and type of surgery
    b. Is extremely poor for patients who suffer from severe CVAs

VII. Initiation of Mechanical Ventilation

A. Criteria for establishing respiratory failure
    1. Neuromuscular depression or dysfunction
    2. Chest wall injury or dysfunction
    3. Airway obstruction
    4. Alveolar filling abnormalities
    5. Increased V/Q abnormalities
    6. Decreased V/Q abnormalities
B. Establishment of an airway
    1. Oral or nasal insertion of endotracheal tube
    2. During procedure, assist patient with bag-mask devices and supplemental oxygen
    3. Auscultate and verify tube position with x-ray
    4. Secure with tape, inflate cuff
C. Establishment of initial ventilator parameters
    1. Blood gas values
    2. Clinical condition and disease process
    3. Tidal volume is established according to body weight
    4. Flow rate adjusted 1:1.5
    5. Tidal volume and sigh rate pressure limits are set 10-20 cmH2O above peak cycling pressures during delivery
    6. Alarms for ventilator failure, low-pressure and/or disconnect, low tidal volume delivery
    7. If patient "fights" the ventilator, sedate

VIII. Weaning From Mechanical Ventilation

A. Methods of evaluation and criteria
    1. See Table 4 for summary

B. Arterial blood gas values
    1. Normal acid-base status
    2. PaO2 greater than 70 mmHg on 40% oxygen
        a. For COPD patient, a return to prior values

C. Alveolar-arterial oxygen gradient
   1. P(A-a)O2 less than 300 mmHg on 100% oxygen is adequate
D. Peak inspiratory pressure
   1. PIP greater than 20 cmH2O is adequate
E. Minute ventilation
   1. Less than 10 lpm
F. Maximal voluntary ventilation
   1. Patient should be able to double their resting minute ventilation
G. Respiratory rate
   1. Less than 30 bpm
H. Vital capacity
   1. Greater than 10-15 ml/kg body weight
I. Forced expiratory volume in 1 second
   1. At least 10 ml/kg body weight
J. Tidal volume
   1. Greater than 200 ml
K. VD/VT ratio
   1. Less than 0.55
L. Shunt fraction
   1. Less than 15%
M. Other factors
   1. Psychological preparation
   2. Cardiovascular status
   3. Nutrition
   4. Fatigue
   5. Fluid balance

IX. Methods Of Weaning

A. Ventilator discontinuance
B. Spontaneous breathing trials
C. SIMV
D. Pressure support ventilation
E. AMV (bears)

X. Nutritional Support

A. Factors contributing to malnutrition
    1. Inadequate intake, particularly calories and protein
    2. Inadequate absorption
    3. Decreased utilization
    4. Increased losses of nutrients
    5. Increased requirements
B. Methods of nutritional support
    1. Entail
    2. Parenteral
C. Evaluation of nutritional support
    1. Body weight
        a. Fats are converted first, then protein
        b. Even the obese can suffer protein-caloric malnutrition
D. Nutritional support in the ventilator patient
    1. Muscle weakness and atrophy
        a. From disuse
        b. From protein breakdown for energy needs
    2. Excessive glucose administration in the malnourished ventilator patient increases respiratory quotient
    3. Closely monitor weight loss
        a. Serial determinations of arm muscle circumference
        b. Indirect calorimetry using a metabolic chart is mostly reliable

SECTION II - INTRA-AORTIC BALLOON PUMP

LEARNING OBJECTIVES

Upon completion of this section, given an open-book, multiple-choice exam, you will be able to apply the information learned to correctly answer a minimum of 90% of the test items. Successful completion of this exam will require you to have mastered the following learning objectives:

1. List four clinical applications of the intra-aortic balloon pump.

2. Describe the location of the balloon.

3. Describe the synchronization of the balloon with the cardiac cycle and how the balloon inflation and deflation is paced.

4. Identify the physiological monitors necessary during intra-aortic balloon pumping.

5. Identify the primary physiologic effects of intra-aortic balloon pumping.

6. Describe the complications and side effects associated with intra-aortic balloon pumping.

7. Indicate the contraindications for intra-aortic balloon pumping.

8. Describe the procedure for weaning the patient from the intra-aortic balloon pump.

INDICATION FOR IABP

The intra-aortic balloon pump (IABP) is a safe and effective device for temporarily augmenting inadequate circulation due to a failing heart. The IABP is normally continued until the function of the heart can be restored either pharmacologically or surgically.

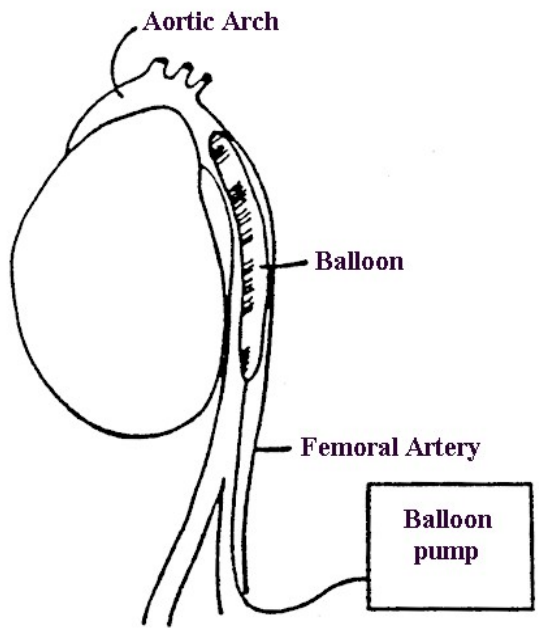
The IABP can be used successfully to augment circulation in 1) post-cardiopulmonary by-pass, 2) cardiogenic shock, 3) myocardial infarction, and 4) refractory cardiac ischemia. IABP is particularly useful following cardiac surgery as a means of removing the patient from cardiopulmonary by-pass when the patient's cardiac output is marginal. In this situation the patient is placed on the balloon pump in surgery prior to transfer to the recovery/intensive care area.

Operation of the IABP Devices

The balloon is attached to a catheter, which is passed into the descending thoracic aorta via a femoral arteriotomy or percutaneous puncture. The balloon may have one or three segments. The three-segment balloon is designed so that the middle segment inflates before the two other segments. The tip of the balloon is positioned just distal to the left subclavian artery (Figure 1).

Figure 1

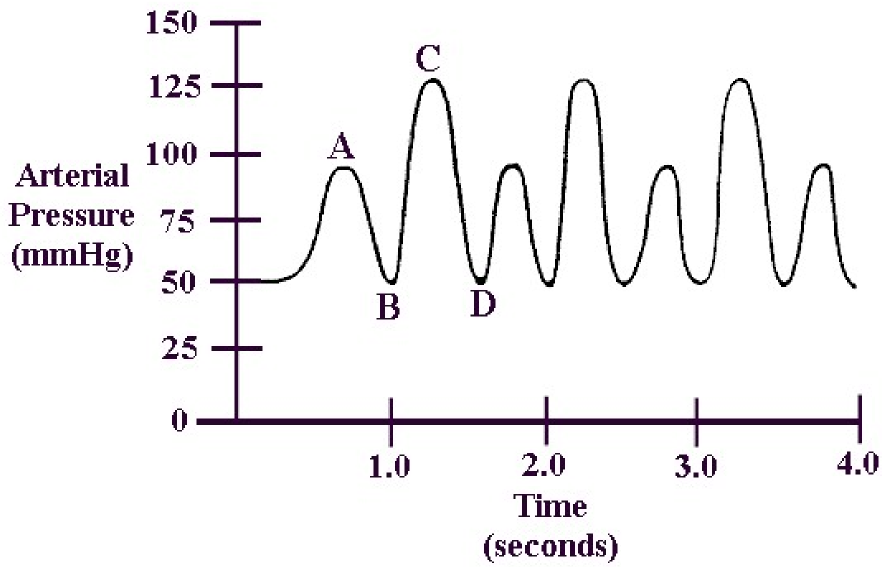
Position of the Intra-aortic Balloon



The alternating inflation and deflation of the balloon is synchronized with the cardiac cycle. Balloon inflation occurs during diastole while deflation occurs during systole. The R-wave of the ECG is normally used as the triggering mechanism for synchronizing the deflation and inflation of the balloon with the systolic and diastolic phases of the heart. A high quality ECG output must be maintained in

order to assure proper sensing of the ECG wave pattern. It is customary to select the lead that produces the largest R-wave. Besides determining the triggering mechanism, the ratio and balloon volume can also be varied on the IABP device. Initially the ratio is set at 1:1 so that each contraction of the heart is followed by an inflation of the balloon (Figure 2). Initially, the balloon volume is usually set on maximum. As the patient's cardiac status improves, the ratio is gradually reduced to wean the patient from the IABP. On most devices the ratio can be reduced to 1:8, where there will be one balloon inflation for every eight heart contractions.

Figure 2
IABP Pressure Wave Form



A = Peak systolic pressure due to normal left ventricular contractions

B = Initial balloon inflation

C = Peak Balloon inflation/peak siastolic pressure

D = Balloon deflation

An arterial line is required to monitor the pressure wave forms produced by the heart contractions and the IABP device. This will confirm that the IABP is synchronized with the heart cycle and will monitor the effectiveness of the procedure. Either helium or carbon dioxide is used as the balloon-inflating gas; however, the lower viscosity of helium allows the gas to be pumped more rapidly into and out of the balloon, making possible the higher inflation rates needed in patients having tachycardia.

Physiological Effects

The primary physiological effect of IABP is augmentation of the pumping action of the heart resulting in an increase in the mean arterial pressure and improvement of the coronary perfusion. This augmentation of the arterial pressure reduces the left ventricular work. The myocardium benefits in two ways. First, the reduction in the cardiac work results in a decreased oxygen demand by the myocardium and second, the increased coronary artery perfusion increases oxygen supply to the myocardium.

Complications and Side Effects

IABP generally has minimal side effects and hazards. Damage to the aorta and blood constituents is minimal. Complications include wound infections, thrombocytopenia, thrombus formation and embolization. Although infrequent, trauma to or dissection of the aorta can occur. Finally, as with any piece of medical equipment, malfunctions can occur in the operation of the IABP device.

Contraindications

IABP is contraindicated where there is extensive peripheral vascular disease, which prevents the insertion of the balloon. Also, IABP should not be used when aortic regurgitation is present or possible.

Weaning/Discontinuance

As mentioned above, normally the patient is initially established on a 1:1 ratio. As the patient's cardiovascular status improves, the ratio is gradually reduced to 1:8. At this point discontinuance of the IABP is normally possible. Prior to discontinuing IABP, several indices should be evaluated. These include the cardiac output, mean arterial pressure, pulmonary capillary wedge pressure, and arterial pressure, pulmonary capillary wedge pressure, and arterial oxygen tension with the IABP off. The balloon is removed only after the patient demonstrates cardiovascular stability for several hours without assistance from the balloon pump.

STUDY OUTLINE

I. Indications for IABP

   A. Purpose is to temporarily augment inadequate circulation due to a failing heart
   B. Indicated for:
      1. Post-cardiopulmonary bypass
      2. Cardiogenic shock
      3. Myocardial infarction
      4. Refractory cardiac ischemia

II. Operation of the IABP Device

   A. Balloon is attached to a catheter, passed into the descending thoracic aorta via femoral arteriotomy or percutaneous puncture
      1. Balloon may have one or three segments
   B. Balloon inflation and deflation is synchronized with the cardiac cycle
      1. Inflation occurs during diastole, deflation during systole
      2. The R-wave is the triggering mechanism
      3. A high-quality ECG must be maintained
   C. The ratio and balloon volumes can be varied
      1. Ratio is initially set at 1:1, but can be reduced to 1:8 during weaning
   D. An arterial line monitors the pressure waves
      1. Confirms synchronization of IABP and heart cycle
      2. Monitors effectiveness
   E. The balloon-inflating gas is either helium or carbon dioxide
      1. Lower-viscosity helium can be pumped more rapidly, is favored for patients with tachycardia

III. Physiological Effects

   A. The augmentation of the heart's pumping action increases mean arterial pressure and improves coronary perfusion
      1. Aid to arterial pressure reduces left ventricular work
   B. Myocardium benefits
      1. Decreased workload reduces oxygen demands
      4. Increased coronary artery perfusion increases oxygen supply to the myocardium

IV. Complications and Side Effects

   A. Damage to the aorta and blood constituents is minimal
   B. Complications
      1. Wound infections
      2. Thrombocytopenia
      3. Thrombus formation
      4. Embolization
   C. Trauma or dissection of the aorta
   D. Equipment malfunctions

V. Contraindications

A. When there is extensive peripheral vascular disease preventing balloon insertion
B. When aortic regurgitation is present or possible

VI. Weaning/Discontinuance

A. The ratio is gradually reduced to 1:8
B. Check these indices with the IABP off:
    1. Cardiac output
    2. Mean arterial pressure
    3. Pulmonary capillary wedge pressure
    4. Arterial oxygen tension
C. The balloon is removed only after the patient demonstrates cardiac stability for several hours without the balloon pump's assistance

SECTION III - EXTRACORPOREAL MEMBRANE OXYGENATION

LEARNING OBJECTIVES

Upon completion of this section, given an open-book, multiple-choice exam, you will be able to apply the information learned to correctly answer a minimum of 90% of the test items. Successful completion of this exam will require you to have mastered the following learning objectives:

1. Describe the clinical situation in which extracorporeal membrane oxygenation might be indicated.

2. Indicate the two primary physiological benefits of extracorporeal membrane oxygenation.

3. Describe the two primary vessel routes for administering extracorporeal oxygenation.

4. Indicate the advantages and disadvantages of the two routes for administrating extracorporeal membrane oxygenation.

5. Describe other facets or care related to maintaining the patient on extracorporeal membrane oxygenation including:

    a. heparinization
    b. ventilator management
    c. secretion evacuation

6. Describe the desirable physiological effects of extracorporeal membrane oxygenation

7. Describe the adverse physiological effects of extracorporeal membrane oxygenation

8. Indicate the kinds of physiologic monitors required to properly manage the patientreceiving extracorporeal membrane oxygenation.

9. Indicate the complications associated with extracorporeal membrane oxygenation.

10 Describe the procedure for weaning the patient for extracorporeal membrane oxygenation.

INDICATIONS AND OBJECTIVE FOR ECMO

Simply put, extracorporeal membrane oxygenation (ECMO) is heart-lung bypass. The technique of cardiopulmonary bypass developed for open heart surgery is expanded to permit long-term support over several days. It is indicated for the patient in severe acute respiratory failure who does not respond to maximum methods of conventional care. These methods would include endotracheal intubation or tracheotomy, mechanical ventilation, high PEEP levels, high oxygen concentrations, diuresis, airway suctioning, chest physiotherapy, and other appropriate medical procedures.

Extracorporeal oxygenation has two primary objectives. First and foremost, oxygenation is significantly improved which reduces the danger of oxygen toxicity due to high ventilator oxygen concentrations. At the same time, the PEEP level, peak airway pressure, and mean airway pressure can be reduced, which lowers the intrathoracic pressures. This overall reduction in the intrathoracic pressures results in less barotrauma damage to the lungs and improves the cardiac status by increasing venous return to the right heart. Second, extracorporeal oxygenation "buys" time by allowing the lungs to return to their normal physiologic function. At the same time, the overall internal environment of the body is improved during ECMO because of the improved oxygenation. As a result, the patient's chances of complete recovery are increased..

The decision to administer extracorporeal oxygenation should be made early in the course of the disease before there is severe damage to the lungs from high ventilating pressures and high concentrations of oxygenation. If the disease is allowed to progress to the point where pulmonary gas exchange is severely compromised and the chest x-ray shows complete opacification for more than 12 hours, the patient's prognosis becomes very poor even after the implementation of extracorporeal oxygenation.

CANNULA SITES FOR ECMO

There are two major routes for administering ECMO, the venovenous and venoarterial. The advantages and disadvantages are discussed below.

The venovenous route has a cannula placed in the inferior vena cava via the femoral vein, and another cannula placed in the superior vena cava via the internal jugular vein (Figure 1). Two incisions are required for the venovenous method: one in the femoral vein and the other in the internal jugular vein. Venous blood from the inferior vena cava is directed to the ECMO where it is oxygenated and returned to the body via the superior vena cava.

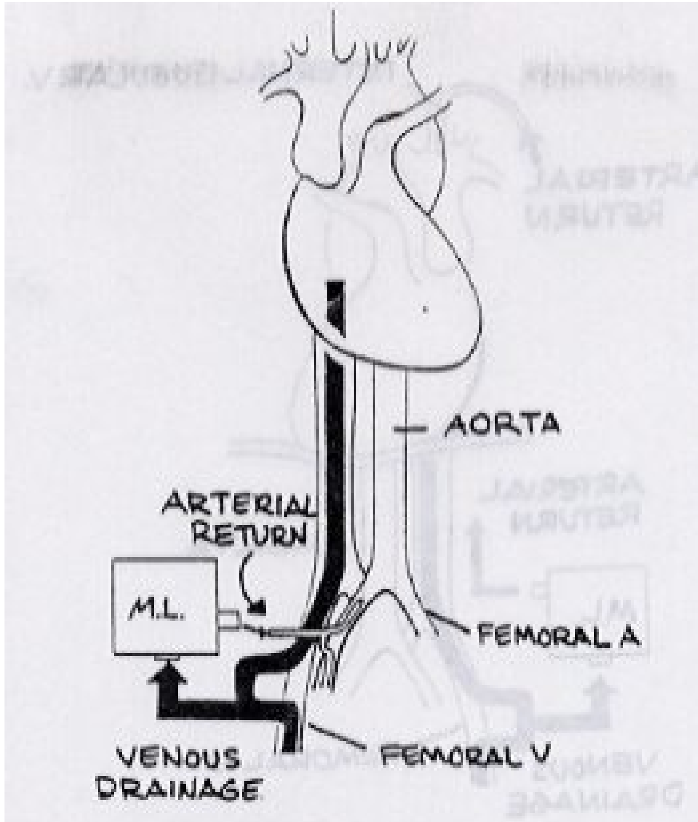


Figure 2

Venoarterial Route

The primary advantage of the venovenous method is that it produces a uniform distribution of the arterialized blood throughout the body. Specifically, the lower and upper parts of the body equally receive the arterialized blood. A major disadvantage of the venovenous method is that all of the blood volume from the ECMO flows through the lungs. This has the undesirable effect of increasing the pulmonary blood flow and increasing the load on the right ventricle. In the face of pre-existing right heart failure, this increase in blood flow can result in pulmonary hypertension and edema. Finally, a relatively low maximum by-pass blood flow of 50 percent of the cardiac output can be obtained using the venovenous route to seal the vessels. With balloon cannulas, up to 80-90 percent of the cardiac output can be diverted to the ECMO, improving the oxygenating capability.

The second method for administering ECMO is the venoarterial route, which uses the femoral vessels only. One cannula is placed in the inferior vena cava via the femoral vein and another in the common femoral artery (Figure 2). Venous blood from the inferior vena cava is directed to the ECMO where it is oxygenated and returned to the body via the femoral artery. Two incisions are required: one in a femoral vein and the other in a femoral artery.

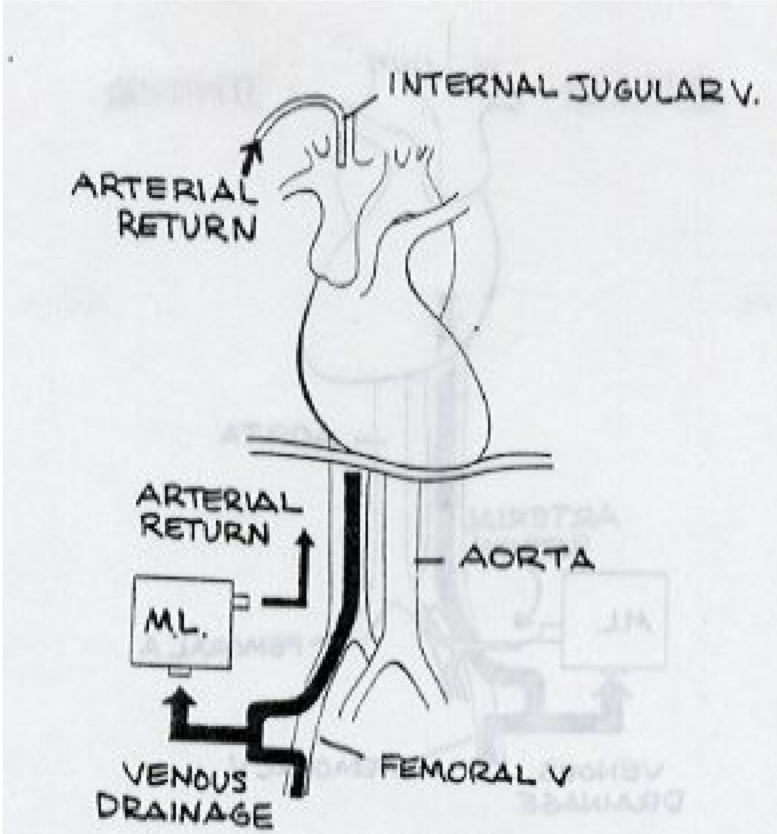


Figure 1

Venovenous Route

The major advantage of the venoarterial route is that maximum by-pass blood flows of 60-70 percent of the cardiac output can be achieved without balloon cannulas. A second advantage of the venoarterial route is that blood flow through the lungs is reduced since the oxygenator blood flow is not directed through the right side of the heart. This results in a reduction in the work required of the right heart, reducing the pulmonary arterial pressures. In other words, the advantage of the venoarterial route is that the pulmonary blood flow, pulmonary artery pressure, and pulmonary resistance are all significantly reduced. This assists in improving the patient's prognosis. A disadvantage of the venoarterial route is the fact that the location of the arterial cannula in the femoral artery results in poor distribution of the arterialized blood in the body. Arterialized blood returning from the oxygenator is distributed preferentially to the lower half of the body. As a result, the head and heart, in particular, are not oxygenated as well. Inserting an additional cannula into the superior vena cava via the internal jugular vein can modify this situation. This cannula directs a supply of arterialized blood from the oxygenator to the head and heart. Another method of modifying the venoarterial route to improve delivery of arterialized blood to the head and heart involves inserting the arterial return cannula into the aortic arch with the tip located at the level of the subclavian artery.

MAINTENANCE OF ECMO

Passage of the blood through the ECMO increases the chances of thrombotic phenomena in the patient. As a result, the patient must be heparinized. Clotting time is monitored frequently to maintain an appropriate level of heparinization, since over-heparinization can result in uncontrollable bleeding. During ECMO the ventilator oxygenation concentration should be set at 50% and the PEEP at 5 cmH_2O. Adequate airway humidification, airway suctioning, and chest physiotherapy should be maintained in order to promote healing of the lungs while the patient is on ECMO.

PHYSIOLOGICAL EFFECTS OF ECMO

The primary physiological effect of ECMO is an improvement in oxygenation. Adverse affects include a significant fall in the platelet count when extracorporeal oxygenation is initiated. The white cell count also falls, but usually returns to normal within a short period of time. The effects on the cardiovascular system depend somewhat on the route of cannulation used, as discussed above.

MONITORING OF ECMO

Frequent arterial blood gases should be taken to monitor oxygenation and acid-base balance. The patient's acid-base balance will still depend on the appropriate delivery of ventilation via the ventilator. The patient should have a Swan-Ganz line to facilitate the measurement of the cardiac output, pulmonary wedge pressure, and pulmonary artery pressure. Breath sounds should be monitored closely for signs of improvement or further deterioration. Daily chest x-rays should be taken to monitor the progress of the disease. An x-ray that gradually becomes more opacified while the patient is on ECMO is a poor sign. On the other hand, an improvement in the chest x-ray is normally associated with an improvement in pulmonary gas exchange.

COMPLICATIONS OF ECMO

Hemorrhaging as a result of the anticoagulation therapy is a common complication of prolonged ECMO. As indicated above, clotting time is monitored closely to reduce this hazard.

Sepsis is a major complication in prolonged use of ECMO. Frequent blood cultures should be taken to monitor this aspect of the patient's care and appropriate antibiotics administered. Obviously, meticulous care of the ECMO equipment, cannulas, and incision sites is mandatory.

Disseminated intravascular coagulation (DIC) is another complication that can occur during ECMO. However, it should be noted that DIC is a phenomena that can occur in any critically ill patient. The occurrence of DIC is not caused by ECMO.

WEANING FROM ECMO

The criteria for weaning involves an examination of the effect of reduced oxygenator blood flow on arterial oxygenation. If the arterial oxygenation remains relatively stable as the oxygenator blood flow is reduced, weaning from the oxygenator can be initiated. Mechanical ventilation with 50% oxygen and 5 cmH_2O PEEP is continued during the weaning procedure.

If a PaO_2 greater than 50 mmHg is maintained for several hours after the oxygenator flow is terminated, ECMO can be discontinued; however, during the weaning process, the oxygenator blood flow should never be stopped completely, as this can result in thrombosis. A minimal blood flow should be maintained via the oxygenator until the cannulas are removed.

STUDY OUTLINE

I. Indications for ECMO

A. Indicated for patient in severe acute respiratory failure who does not respond to maximum conventional care
   1. Conventional care includes:
      a. Endotracheal intubation or tracheotomy
      b. Mechanical ventilation
      c. High PEEP levels
      d. High oxygen concentrations
      e. Diuresis
      f. Airway suctioning
      g. Chest physiotherapy

B. Objectives
   1. To improve oxygenation, reducing the risk of oxygen toxicity from high ventilator oxygen concentrations
      a. PEEP level, peak airway pressure, mean airway pressure can be reduced, lowering the intrathoracic pressures
      b. Causes less barotrauma to lungs, and increases venous return to the right heart
   2. To gain time, allowing the lungs to return to their normal physiologic function
      a. Overall internal environment is improved, increasing the patient's change of complete recovery
   3. The decision to use ECMO should be made early in treatment, before severe damage to the lungs has occurred

II. Cannula Sites For ECMO

A. Venovenous route
   1. Placement of a cannula in the inferior vena cava via the femoral vein, and in the superior vena cava via the internal jugular vein
      a. Venous blood from the inferior vena cava is directed to ECMO for oxygenation
      b. Blood is returned to the body via the superior vena cava
   2. Advantages
      a. Produces a uniform distribution of arterialized blood throughout the body
   3. Disadvantages
      a. All ECMO blood volume flows through the lungs increasing the load on the right ventricle
      d. There is a low maximum by-pass flow of 50% of the cardiac output

B. Venoarterial route
   1. Placement of a cannula in the inferior vena cava via the femoral vein, and another in the common femoral artery
      a. Venous blood from the inferior vena cava is directed to the ECMO
      b. Blood is returned to the body via the femoral artery
   2. Advantages

a. Maximum by-pass blood flows of 60-70% can be achieved without balloon cannulas
b. Blood flow through the lungs is reduced, reducing pulmonary blood flow, pulmonary artery pressure, and pulmonary resistance

3. Disadvantages
a. Location of arterial cannula gives poor arterialized blood distribution
1) Head and heart can get better oxygenation with an additional cannula in the superior vena cava (from internal jugular)
2) An alternate delivery system to head and heart is insertion of a cannula in the aortic arch, with the tip at the level of the subclavian artery

III. Maintenance

A. Heparinization is required, because of increased risk of thrombotic phenomena
1. Calls for frequent monitoring of clotting time

B. Oxygenation concentration set at 50%; PEEP at 5 cmH_2O
1. Maintain adequate airway humidification, suctioning, and chest physiotherapy to promote healing

IV. Physiological Effects of ECMO

A. Primary effect is improved oxygenation
B. Adverse effects
1. Significant fall in platelet count
2. Temporary fall in white blood cell count

V. Monitoring of ECMO

A. Frequent arterial blood gases
1. Acid-base balance depends on the ventilator

B. Swan-Ganz line to facilitate measurement of cardiac output, pulmonary wedge pressures, pulmonary artery pressure
C. Breath sounds
D. Daily chest x-rays
1. Gradual increase in opacity is a poor sign

VI. Complications of ECMO

A. Hemorrhaging as a result of anticoagulation therapy
B. Sepsis
C. Disseminated intravascular coagulation
1. Can occur in any critically ill patient; is not caused by ECMO

VII. Weaning From ECMO

A. Test the effect of reduced oxygenation blood flow on arterial oxygenation
   1. If it remains stable, weaning is indicated
   2. If PaO2 greater than 50 mmHg is maintained for several hours after flow is terminated, ECMO can be discontinued

B. During weaning, do not stop oxygenation blood flow completely until cannulas are removed
   1. Otherwise, thrombosis could occur

SECTION IV - CLOSED-CHEST DRAINAGE

LEARNING OBJECTIVES

Upon completion of this section, given an open-book, multiple-choice exam, you will be able to apply the information learned to correctly answer a minimum of 90% of the test items. Successful completion of this exam will require you to have mastered the following learning objectives:

1. List and describe the anatomical structures in the three compartments in the chest.

2. Describe the anatomical characteristics and function of the visceral pleura, parietal pleura, and the intrapleural space.

3. Define the following terms:

    a. Pneumothorax
    b. Hemothorax
    c. Pyothorax
    d. Chylothorax
    e. Hydrothorax

4. Indicate some of the causes of air entering the pleural space from the atmosphere (outside the lung).

5. Indicate the primary cause of air entering the pleural space from within the lung.

6. Differentiate between a closed pneumothorax and an open pneumothorax.

7. Discuss the pathophysiology, symptoms, hazards, and treatment of a tension pneumothorax.

8. List five indications for chest tubes.

9. Describe the clinical and laboratory picture seen in fluid or air accumulation in the pleural space.

10. Indicate the three major components of a simple closed-chest drainage system. Describe the function of each of the components.

11. Indicate the possible explanations for the lack of fluctuation of the water seal in any closed-chest drainage system.

12. Indicate the average amount of negative pressure that is usually applied to the pleural space in adult patient if suction is used.

13. Describe other procedures that should accompany closed-chest drainage in order to facilitate re-expansion of the lung and adequate secretion evacuation from the airways.

14. Describe the measures that should be taken after a chest tube is removed.

LUNG PHYSIOLOGY

The chest has three compartments: the separate pleural cavities for each lung, and the mediastinum. Each lung surface is covered with a visceral pleura, while the chest wall is covered with a parietal pleura. Normally, these two pleural surfaces are separated by only a thin lubricating pleural fluid, which allows the two pleural surfaces to slide smoothly over one another.

In the normal lung, the pleural space is actually a potential space; however disease, trauma, or surgery can disrupt the space and enable air, fluid, or blood to enter. The accumulation of air, fluid, or blood in the pleural space, if not corrected, can result in complete collapse of the affected lung and severe respiratory distress. Large volumes of air, fluid, or blood should be removed from the pleural space with a closed-chest tube drainage system.

INTRAPLEURAL AIR AND FLUID ACCUMULATION

The following terms describe the kinds of fluid and air collection that can occur in the pleural space:

Pneumothorax - a collection of air in the pleural space

Hemothorax - a collection of blood in the pleural space

Pyothorax - a collection of pus in the pleural space

Chylothorax - a collection of chyle from the thoracic duct
Hydrothorax - a collection of serum, hyperalimentation, or other intravenous fluid from a misplaced central venous catheter

Air can enter the pleural space from inside the lung or from outside. Air that enters the pleural space from the outside usually does so via a surgical incision or penetrating blunt chest trauma. Air that enters the pleural space from inside the lung usually does so via ruptured blebs, for example, in the lungs of COPD patients. Other causes of pneumothoraces include lung biopsies, misplaced central venous lines, thoracentesis, and fractured ribs.

There are two types of pnuemothroaces: open and closed. In an open pneumothorax, air goes into and out of the pleural space as the patient breathes, with very little accumulation. In the closed pneumothorax, a one-way valve mechanism exists wherein air can enter the pleural space but does not exit. As a result, there is an accumulation of air in proportion to the size of the orifice communicating with the pleural space. This can quickly become life-threatening and is referred to as a tension pneumothorax. The increasing accumulation of air in the pleural space results in the collapse of the affected lung. A severe tension pneumothorax can also compress the mediastinum, interfering with venous return to the heart and reducing the cardiac output. The unaffected lung can even become compressed. Obviously, the patient's pulmonary and cardiac function become severely compromised.

Clinical Symptoms

Accumulation of significant amounts of air, fluid, or blood in the pleural space will be accompanied by certain clinical symptoms and laboratory test results.

Early symptoms of a tension pneumothorax include: dyspnea, chest pain, tachypnea, tachycardia, diaphoresis and anxiety. The breath sounds on the affected side will be decreased or absent. There may be accompanying subcutaneous emphysema and tracheal deviation. If the pneumothorax becomes significant, arterial blood gases will show hypoxemia and hypercapnia on low-flow oxygen. A chest x-ray is the definitive diagnostic procedure for determining the presence of air, fluid, or blood in the pleural space. The PaO2 will also be decreased. The presence of air in the pleural space will produce either decreased, absent, or hyper-resonant breath sounds. Fluid or blood in the pleural space will produce distant, dull, or absent breath sounds.

A tension pnuemothorax requires immediate medical intervention if pulmonary failure and circulatory collapse are to be prevented.

Chest Tubes

A chest tube is simply a gravity-driven drain. A closed-chest tube drainage system is designed to drain fluid, blood, or air from the pleural space while at the same time maintaining a negative pressure within the intrapleural space and preventing the return of air or fluid back into the pleural space.

Indications

Chest tubes may be required for 1) three to four days after a lung resection, 2) one to two days after a pneumonectomy, 3) two days after open-heart surgery, 4) in treating hemothoraces, and 5) in treating pneumothoraces. Closed-chest tube drainage is indicated when excess air, fluid, or blood is present in the pleural space and removal by simple thoracentesis will not be adequate. It should be noted that air cannot be removed via a thoracentesis.

Theory of Operation

A closed-chest tube drainage system consists of three components: 1) a chest tube, 2) a collection container, and 3) a water seal. The chest tube permits the flow of fluids and air out of the lung. Chest tubes come in varying sizes. In the adult patient a #28 to #36 French tube is used. Children will

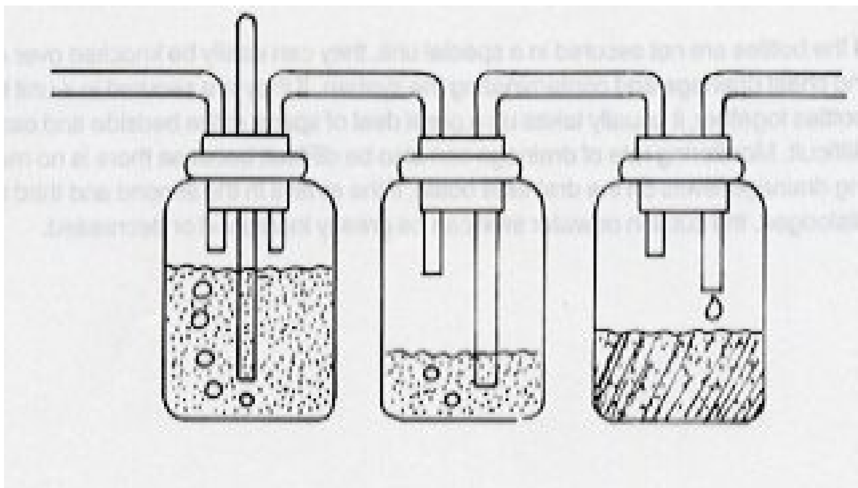
require smaller tubes. The water seal acts as a one way valve, establishing a closed system by sealing off the open end of the chest tube from the atmosphere. Excess air, fluid and blood move out of the pleural space, but atmospheric air is prevented from entering. The drainage bottle should be kept several feet below the patient since gravity determines the flow of the fluid.

TYPES OF CLOSED-CHEST DRAINAGE SYSTEMS

Three-Bottle Chest Drainage Systems

Traditionally, chest drainage was accomplished with a three-bottle chest drainage system. In this system, three interconnected glass bottles are lined up on the floor; each bottle has a separate function (figure 15).

Figure 15



Three-bottle System

Collection Bottle

First is the collection bottle. Often referred to as "bottle one", this bottle is nearest the patient. Drainage enters the bottle from chest drainage tubing through a shortened straw at its top.

Water Seal Bottle

The second bottle, "bottle two" is the water seal bottle. Under normal conditions, negative pressure in the pleural cavity bolds the pleurae together and keeps the lung up against the chest wall. If air or fluid gets into the pleural cavity, the negative pressure is disrupted, compromising full expansion of the lung. The water seal acts as a one-way valve; it allows air to leave the pleural cavity, but not to return, thus maintaining negative pressure.

Think of it as blowing bubbles through a soda straw-you can blow the bubbles through the straw and soda, but when you draw through the straw, just fluid-not air-comes up. The drainage straw should be placed under 2-3cm of water to make an effective seal.

Suction Control Bottle

The third bottle, "bottle 3", is the suction control bottle. Essentially a safety device, it limits the amount of suction that can be applied to the pleural cavity, regardless of the amount set on the regulator.

The bottle contains water and a straw. The amount of suction is regulated by how deep the straw is submerged. The top of the straw is open to the atmosphere. The regulated suction device will apply suction through the entire system until it reaches the pressure that will draw atmospheric air in through the open straw. At this point, any increase in suction will be used to draw in more atmospheric air and will not be transmitted to the pleural cavity.

In a three-bottle system, it is the depth of the straw in the suction control bottle that regulate the suction; as long as the water is bubbling, increasing the suction on the regulator will increase the air flow through the system only, not the suction pressure on the pleural cavity.

Three-bottle systems do a good job at maintaining chest drainage, but they do have their drawbacks. One is most apparent during set-up. Numerous parts and connections make the system difficult to assemble and dangerous errors can occur. Handling the parts also makes contamination likely and increases the risk of air leaks developing between the bottles. The bottles themselves are very heavy and cumbersome, which makes transporting the patient with a chest drainage system difficult. The water in the second bottle can slosh, and the seal can be lost, allowing reaccumulation of the pneumothorax.

If the bottles are not secured in a special unit, they can easily be knocked over and broken, disrupting chest drainage and contaminating the system. If they are secured in a unit that holds all three bottles together, it usually takes up a great deal of space at the bedside and can make patient care difficult. Monitoring rate of drainage can also be difficult because there is no mechanism for marking drainage levels on the drainage bottle. If the straws in the second and third bottles become dislodged, the suction or water seal can be greatly increased or decreased.

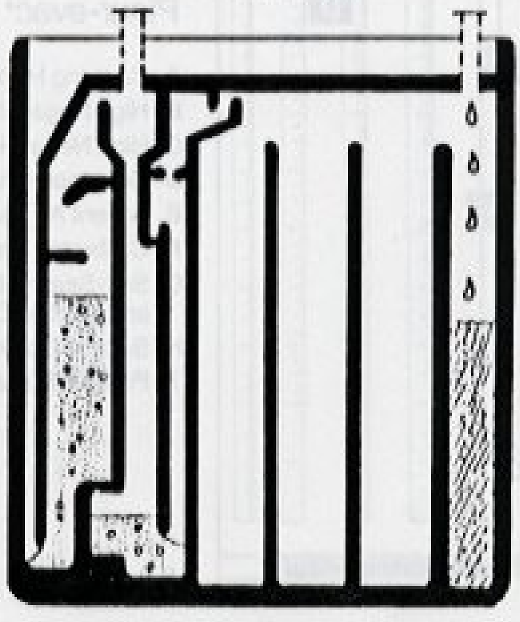
The Pleur-evac Chest Drainage System

Collection Chamber

The Pleur-evac takes the traditional functions of the three-bottle system and integrates them into one plastic, disposable unit. As you look at the Pleur-evac, you can see how the chambers correlate to the three bottles in the traditional systems (figure 16). At the right side of the unit is the collection chamber, which correlates to "bottle 1". The 6 foot (2m) tubing connects this part of the Pleur-evac directly to the chest tube. Any fluid drainage form the chest goes into this chamber. It is calibrated in 1 ml increments up to 100 ml, 2 ml increments from 100 ml to 200 ml and 5 ml increments from 200 ml to 2500 ml. It has surface on which you can mark the time an date of drainage.

Figure 16

Three-bottle System



Pleur-evac® A-7000

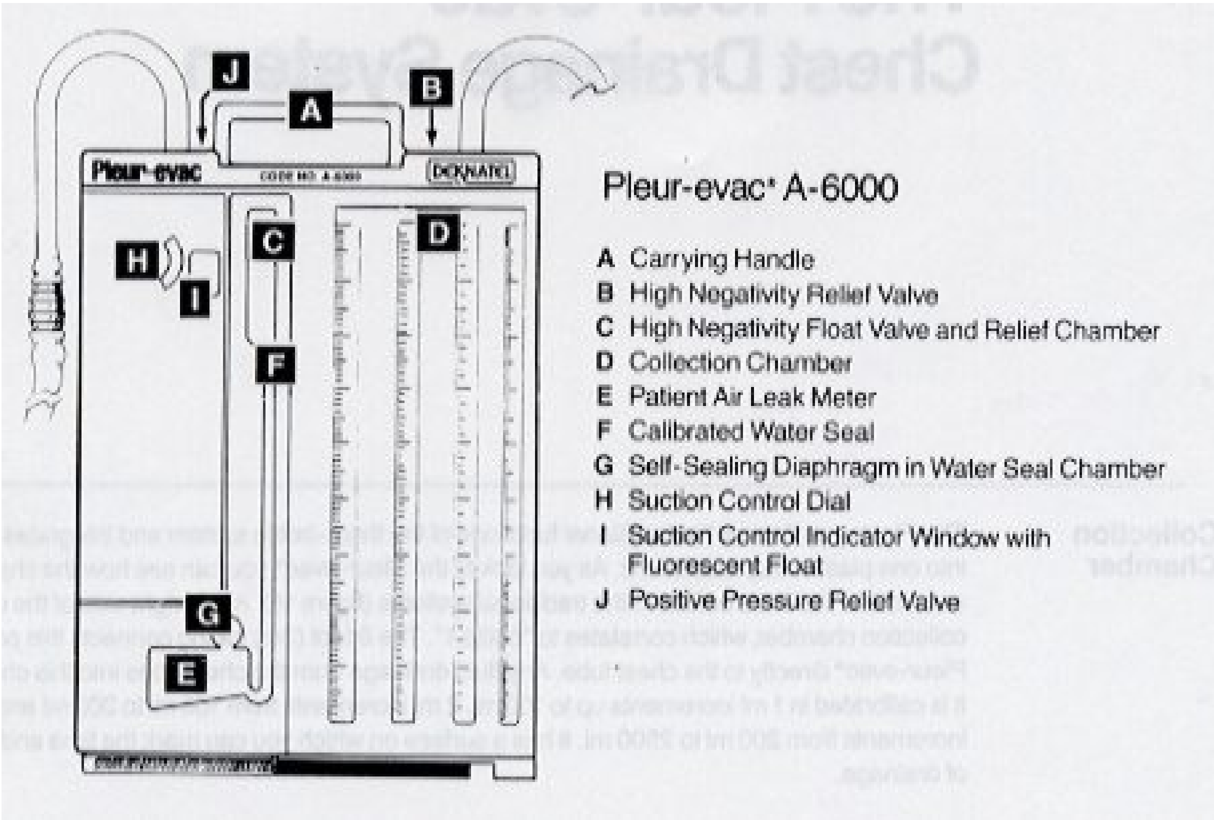
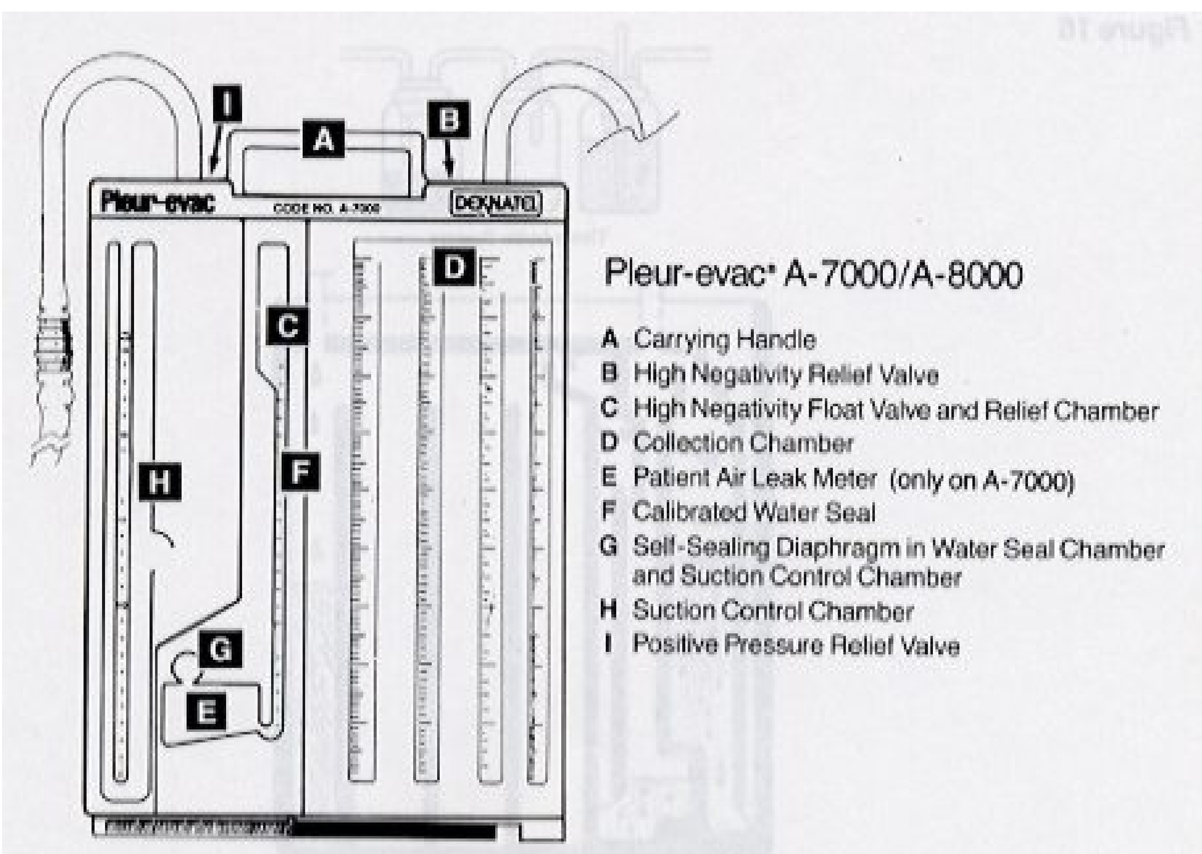

Pleur-evac
CODE NO. A-6000
DEKNATEL
Pleur-evac* A-6000
A Carrying Handle
B High Negativity Relief Valve
C High Negativity Float Valve and Relief Chamber
D Collection Chamber
E Patient Air Leak Meter
F Calibrated Water Seal
G Self-Sealing Diaphragm in Water Seal Chamber
H Suction Control Dial
I Suction Control Indicator Window with Fluorescent Float
J Positive Pressure Relief Valve

Figure 17


Pleur-evac
CODE NO. A-7000
DEKNATEL
Pleur-evac* A-7000/A-8000
A Carrying Handle
B High Negativity Relief Valve
C High Negativity Float Valve and Relief Chamber
D Collection Chamber
E Patient Air Leak Meter (only on A-7000)
F Calibrated Water Seal
G Self-Sealing Diaphragm in Water Seal Chamber and Suction Control Chamber
H Suction Control Chamber
I Positive Pressure Relief Valve

Water Seal Chamber

The middle chamber in the Pleur-evac is the water seal chamber, which correlates to “bottle 2” in the traditional system. When this chamber is filled with fluid up to the 2 cm line, a 2 cm H2O water seal is established without need for the cumbersome submerged straw. In addition to maintaining the original purpose of the water seal-keeping air from entering the pleural cavity-the Pleur-evac also has a calibrated manometer in the water seal chamber to measure the amount of negative pressure referred from the pleural cavity. The water level in the water seal manometer rises as intrapleural pressure becomes more negative.

The water level in the water seal should be monitored routinely to check for evaporation. The self-sealing diaphragm on the front of the water seal allows you to add water should evaporation occur. The diaphragm can also be used to withdraw water if the water seal chamber is overfilled.

The Pleur-evac also has a high negativity float valve in the top of the water seal chamber (figure 17). This maintains the water seal in the event of high negative intrapleural pressures, as may occur with the deep breath taken before vigorous coughing, or with forced inspiration from an upper airway obstruction. High negativity can also occur if the chest tubing is stripped. The Pleur-evac chest drainage unit allows you to vent excess negative pressure. This high negativity is indicated by rising water in the water seal chamber. Depressing the high negativity relief valve will allow filtered air into the system. Relieving negativity and allowing the water level to return to baseline in the water seal. In instances of falsely imposed high negative pressure, such as stripping chest tubes, water will continue to rise, filling the high negativity relief chamber at the top of the water seal chamber. This relief chamber will automatically vent excessive negative pressure which will prevent respiratory compromise from accumulated negativity. Water spillover into the collection chamber is also minimized.

The system also has a positive pressure relief valve. It remains closed when suction is applied to the system, but opens whenever pressure within the system becomes positive. Since the only way for air to leave the Pleur-evac is through the suction port, obstruction of the suction line (by rolling the bed on top of the tubing, for instance) could cause accumulation of air in the system leading to tension pneumothorax. This safety feature, not present in glass bottles systems, allows venting of the positive pressure, minimizing the risk of a tension pneumothorax.

The patient air leak meter is made up of numbered columns, reading from 1 (low) to 7 (high). As air flow through the system increases, bubbling will occur toward the higher end of the scale. Decreasing flow will result in bubbling on the lower end of the scale. This feature provides an indication of air lead magnitude, allowing the clinician to monitor air lead increase or decrease as therapeutic interventions (such as adding or increasing PEEP) are made.

Suction Control Chamber

The chamber on the left side of the unit is "bottle 3", or the suction control chamber. The Pleur-evac unit comes with one of two mechanisms to regulate the amount of suction transmitted to the pleural space: wet or dry suction.

The wet suction mechanism regulates the amount of suction by the height of a column of water in the suction control chamber. Note, it's the height of water, not the setting of the suction source, that actually limits the amount of suction transmitted to the pleural cavity. A suction pressure of –20cm H2O is commonly recommended, but lower levels may be indicated for infants and for patients with friable lung tissue, or if ordered by the physician.

To use the wet suction control chamber in the A-7000/A-8000 series, fill the unit with sterile fluid to the desired height. Connect the tubing supplied with the unit to suction tubing, and then to a suction source. Adjust the source suction to produce gentle bubbling in the suction control chamber. The appearance of gentle bubbling assures you that the amount of suction set (by the height of the column of water) is the amount of suction being applied to the chest cavity; excess suction is vented through the bubbling. Increasing suction at the suction source will increase air flow through the system; it will have a minimal effect on the level of suction imposed on the chest cavity.

Excessive source suction will not only cause loud bubbling (which can disturb patients and caregivers); but will also hasten evaporation of water from the suction control chamber; decreasing the suction applied to the chest cavity. Self-sealing diaphragms are provided to adjust the water level in this chamber should overfilling or evaporation occur.

The dry suction control chamber in the A-6000 series is even easier to use. Instead of regulating the level of suction with a column of water, suction is controlled by a self-compensating regulator. A dial on the side of the suction control chamber allows you to set the desired level of suction. Simply turn the dial and watch the orange stripe on the front of the unit. The stripe is analogous to the level of water in the wet suction; it tells you the maximum level of suction desired in the chest cavity.

As with the wet unit, connect the short tubing supplied with the unit to suction tubing, and then to a suction source. Make sure the source provides a minimum of 20 LPM of air flow. Once connected to suction, increase the level of suction until the fluorescent orange float appears in the suction indicator window. This visual confirmation of suction pressure provides the same assurance as the gentle bubbling in the wet suction control chamber. When the fluorescent orange float appears, you are assured that the level of suction set on the dial is being transmitted to the chest cavity.

The Pleur-evac unit provides a unique dry suction design that immediately responds to changes in patient pressure (patient air leak) or changes in suction pressure (surge/decrease at the suction source or an inadvertent change in the suction source). This safety feature assures that the amount of suction set will continue to be the amount transmitted to the pleural cavity, as long as the fluorescent orange float remains in the indicator window. This dry suction safety feature is unique to Pleur-evac. Unlike wet suction systems, this dry suction system can accommodate suction flows as high as 60 LPM and can impose suction pressures as high as –40cm H2O.

With the dry suction unit, the level of suction set can be increased at any time. Simply turn the dial counterclockwise and the orange stripe will confirm the new setting. You may need to increase source suction to keep the fluorescent orange float in the indicator window.

To decrease the level of suction, turn the dial clockwise until the desired level of suction is indicated by the orange stripe. Then look at the water seal. In the absence of a patient air leak, the water in the water seal chamber will rise as the suction setting is decreased. This higher water level indicates the previous higher suction setting. Simply relieve this negativity with the high negativity relief valve until the water reaches the baseline in the water seal chamber. Once the water seal reads "zero", and the fluorescent orange float remains in the indicator window, the level of suction indicated by the orange stripe on the front of the suction control chamber is the amount of suction applied to the chest cavity. (for more information on understanding Pleur-evac dry suction control, please call your local Deknatel sales representative at 1-800-843-8600.)

Not all patients require suction. Suction may be discontinued to transport a patient; it may be discontinued 24 hours before chest tube removal. Hospital policy will state whether an order is needed to institute or discontinue suction. If suctions is discontinued, make sure the suction tubing remains open to atmosphere to allow air to leave the drainage system.

Pleur-evac Advantages

The Pleur-evac offers a number of advantages over the glass three-bottle chest drainage system. Most important, it provides a number of safety features. Because it is one unit, connections don't need to be made between bottles; and straws don't have to be positioned at a specific depth. The Pleur-evac has several safety features described earlier: the high negativity float valve which maintains the water seal, the high negativity relief valve and relief chamber that allows you to vent excess negative pressure in the system; and the positive pressure relief valve that minimizes the risk of a tension pneumothorax. The Pleur-evac is small and lightweight. All units contain a locking floorstand for greater stability and a carrying handle for convenient patient transport or ambulation. Since the Pleur-evac has locking hooks, it can be hung out of the way at the foot of the bed, making it less likely to interfere with bedside care.

The calibrated volumes on the collection chamber allow for careful, accurate monitoring of chest drainage. The write-on surface allows drainage levels to be marked at time intervals, so you can see how much drainage has occurred in any elapsed time frame. If specimens are needed for laboratory analysis, sampling port on all Pleur-evac autotransfusion capable units allows you to withdraw a specimen from the drainage tubing. Units not used for autotransfusion have a self-sealing diaphragm in the collection chamber for specimen withdrawal. Locking connectors which secure the connections from the patient to the autotransfusion unit are also available on all autotransfusion Pleur-evac capable unit.

Set-Up Review

To set up the Pleur-evac, first unwrap it and set it upright. Next, fill the water seal by pouring sterile fluid through the latex suction tubing. It should be filled to the 2cm H2O mark. With wet suction units, fill the suction control chamber through the atmospheric vent. With dry suction units, turn the dial on the side of the suction control chamber so the orange stripe indicates the level of suction desired. Then maintaining sterile technique, connect the latex tubing from the collection chamber to the chest tube. Now, connect the suction tubing to the suction source. Finally, adjust the suction source until you see gentle bubbling in the wet suction control chamber or until the fluorescent orange float appears in the suction indicator window of the dry unit.

Nursing Assessment

Pleural Drainage

Signs and symptoms of pneumothorax and pleural effusion were described earlier. See page 5. Even though chest drainage with the Pleur-evac unit has been established, you must still watch for these signs and symptoms, because a disruption in the drainage system can cause them to recur. Your ability to recognize the development of pneumothorax-for instance, if the tubing between the patient's chest tube and Pleur-evac becomes occluded-can save the patient's life. Don't become complacent once the chest tube is in and chest drainage has been established. No system is foolproof.

When you encounter a patient receiving pleural drainage with a Pleur-evac system in place, your initial action should be a thorough respiratory assessment. Your findings will provide a baseline for monitoring changes as your care for the patient, so document them carefully. Once you have done your physical assessment and are satisfied that the patient's respiratory status is satisfactory, turn your attention to the chest tube and Pleur-evac.

Start with the chest tube insertion site. Make sure the dressing is intact, clean, and dry. Follow the tubing from the chest tube to the Pleur-evac, making sure it is patent and has no kinks or leaks. Check that all tubing connections are taped securely. Make sure there are no hanging, dependent loops of tubing that could get caught on anything or, if the loops contain fluid, cause resistance to flow out of the chest. If the patient is awake and aware, tell him how important it is that the tubing isn't kinked or caught on anything.

Next, look at the Pleur-evac unit itself. First, look at the collection chamber. Note the level and assess the character of the drainage; is it bloody, straw-colored, or purulent? What is the rate of drainage? Then, look at the water seal chamber. Is the water level correct at 2 cm? If you see bubbling, it means air is getting into the system. It could mean a leak from the lung, from somewhere in the tubing, or at the chest wall insertion site. Investigate any significant increase or decrease in the bubbling. The air leak meter provides an objective indication of magnitude of air through the system.

If there is no bubbling, the water level should rise and fall with the patient's respirations, reflecting normal pressure changes in the pleural cavity during respiration. During spontaneous respirations, the water level should rise during inhalation and fall during exhalation. If the patient is receiving positive pressure ventilation, the oscillation will be just the opposite-the water level should fall with inhalation and rise with exhalation.

The magnitude of the oscillation will also depend on how stiff the patient's lungs are and how much of the intrapulmonary pressure is transmitted to the pleural cavity. Positive end expiratory pressure (PEEP) may dampen the oscillations. Again, it depends on how stiff the lungs are and how much PEEP is used. Oscillations may be absent if the lung is fully expanded and suction has drawn the lung up against the holes in the chest tubes.

Next look at the suction control chamber. If you're using wet suction, make sure the water level is where it should be as determined by the doctor's order or hospital policy. Water can evaporate from this chamber so the level may drop; refill the chamber as necessary (turn off suction to refill). Make sure the suction source is set so you see gentle bubbling in the suction control chamber. If suction is not being used, or the patient is being transported check to make sure the suction tubing is open to the atmosphere. The tubing should not be capped or clamped, nor should it be left connected to the suction device with the suction source turned off.

If you're using dry suction, make sure the suction level indicated by the orange stripe is where it should be as determined by the doctor's order or hospital policy. Make sure the suction source is adequate so you see the fluorescent orange float in the suction indicator window.

The patient's diagnosis and need for chest drainage will determine how often to repeat your assessment. Check your hospital policy for specific guidelines.

Mediastinal Drainage

If your patient has mediastinal drainage tubes, your assessment will be somewhat different. In addition to a respiratory assessment, a thorough cardiac assessment will be in order. Be especially watchful for signs of cardiac tamponade. As with all patients having chest drainage, you should check dressings and tubing connections, but with this patient your attention must be directed more toward the collection chamber, since the main purpose of mediastinal tubes is to drain fluid from the mediastinum following heart surgery. Monitor the rate of drainage from the mediastinum as frequently as is indicated by the patient's condition.

A patient with only mediastinal tubes should have no bubbling or fluctuations in the water seal chamber, since the tubes are not in contact with the pleural cavity. Bubbling usually indicates either a leak in the tubing or displacement of the chest tube. The water seal chamber should still be monitored for levels of negative pressures. Not only can this negativity pull the water up in the waterseal chamber, but it also puts the patient at risk for mediastinal trauma and graft trauma depending on the precise location of the distal end of the chest tube within the mediastinum.

Suction should be regulated and monitored as it is with pleural drainage.

Nursing Interventions

There are two critical situations that can occur in patients with chest tubes. In pleural cavity drainage, the major hazard is tension pneumothorax. If you detect signs and symptoms of a tension pneumothorax, the most likely cause is obstructed tubing between the water seal chamber and the patient. The Pleur-evac has a positive pressure relief valve in the water seal chamber which allows venting of excess pressure in the pleural cavity, so any blockage causing symptoms will be proximal to that valve, that is, between the Pleur-evac and the patient. You must quickly assess the tubing's patency and notify the doctor immediately. If you cannot find the source of the obstruction, you may want to change the entire Pleur-evac or the doctor may do a needle thoracostomy to vent the pleural pressure and prevent mediastinal shift while he determines the cause of the pneumothorax.

The two critical situations you are likely to encounter in a patient with mediastinal tubes are either sudden hemorrhage or sudden cessation of drainage. Sudden hemorrhaging in a postoperative cardiac patient is likely caused by a ruptured suture line or blown graft. The patient can lose 1,000-1,500 ml of blood in a matter of minutes. Immediately alert the surgeon and prepare for return to the operating room. The other problem, a sudden (not gradual) cessation of drainage can be caused by accumulated clotted blood which has occluded the mediastinal tube. This situation can lead to cardiac tamponade. To keep the tubes patent, or to dislodge clots you may gently milk the tube.

Milk or Strip?

Milking is the term generally used to mean gentle kneading of the tubing. To milk, you alternately compress and release short sections of the tubing between the Pleur-evac and the chest tube which causes momentary bursts of suction within the tubing (figure 18). Stripping is a much more vigorous procedure during which long segments of the tubing are compressed and released. Stripping can cause dangerous high negative pressures. (Reference Duncan/Erickson). Use extreme caution. Your hospital policy will tell you whether routine milking can be used to either remove clots or prevent them from occluding the tube in the first place.

STUDY OUTLINE

I. Lung Physiology

A. Compartments are pleural cavities and mediastinum
   1. A thin lubricating fluid separates Pleural surfaces
   2. There exists a potential space only

II. Intrapleural Air and Fluid Accumulation

A. Terms
   1. Pneumothorax - air in the pleural space
   2. Hemothorax - blood in the pleural space
   3. Pyothorax - pus in the pleural space
   4. Chylothorax - chyle from the thoracic duct
   5. Hydrothorax - serum, hyperalimentation, or other intravenous fluid from a misplaced central venous catheter

B. Types of pnuemothorax
   1. Open
      a. Air moves in and out with little accumulation
   2. Closed
      a. Air enters, but cannot exit
      b. Increasing accumulation leads to collapse

III. Clinical Symptoms

A. Accumulation of significant amounts of air, fluid, or blood causes:
   1. Dyspnea, chest pain, tachypnea, tachycardia, diaphoresis, and anxiety
   2. Breath sounds on affected side are decreased or absent, perhaps with accompanying subcutaneous emphysema and tracheal deviation
   3. Arterial blood gases may show hypoxemia and hypercapnia on low-flow oxygen
   4. A chest x-ray will determine

IV. Chest Tubes

A Indications
   1. For 3-4 days after a lung resection
   2. For 1-2 days after a pnuemonectomy
   3. For 2 days after open-heart surgery
   4. In treating hemothorax
   5. In treating pneumothorax
   6. Whenever simple thoracentesis is not adequate

B. Theory of operations
   1. Chest tube
      a. Comes in different sizes
   2. Collection container

3. Water seal
    a. Acts as a one-way valve
4. Gravity determines the flow of fluid

V. Types of Closed-Chest Drainage Systems

A. One-bottle system
    1. A gallon bottle, a rubber stopper, a venting tube, and a tube for drainage
    2. Fluctuation in the water column reflects depth of inspiration
        a. If there is no movement in the water column
            1) Tubing is kinked
            2) Tubing is obstructed with clots
            3) Tissue is obstructing chest tube tip
            4) Very little fluid or air remains in the pleural space
    3. A disadvantage is that the collected liquid makes further drainage more difficult
B. Two-bottle system
    1. One bottle is the water seal and the other is the collection container
    2. Advantages
        a. Insures a constant pressure
        b. Better observation of drainage fluid volume and characteristics
        c. Simplified fluid disposal
C. Three-bottle system
    1. Additional force is added by a portable suction device
    2. The third bottle serves as a suction control to protect against too much pressure

VI. Patient Care

A. Position
    1. The chest tube should not restrict position
    2. Semi-sitting promotes drainage
    3. Turn the patient frequently
    4. Drainage increases when patient lies on the affected side
    5. The patient will require treatments to re-expand the affected lung
B. Precautions
    1. Report excessive or substantially reduced drainage
        a. Bloody fluid may indicate increased bleeding
        b. Reduced drainage may indicate tube malfunction due to improper position of tube tip or clotting
    2. If chest tube is accidentally pulled out, immediately cover the site with an occlusive dressing to prevent a pnuemothorax
C. Routine care
    1. Frequent auscultation
    2. Note changes in breath sounds
    3. Signs of dyspnea

    4. Encourage frequent coughing, splinting the affected side with a pillow to decrease pain

D. Discontinuing chest tube
    1. A chest x-ray will confirm the integrity of the pleural space
    2. Frequent monitoring of respiratory status

GLOSSARY

Acidemia: An arterial pH below 7.30

Acidosis: A condition characterized by a base deficit

Alkalemia: An arterial pH above 7.50

Auscultation: The act of listening for sounds within the body to evaluate the condition of the heart, blood vessels, lungs, pleura, intestines or other organs. Auscultation may be performed directly, but most commonly a stethoscope is used to determine the frequency, intensity, duration, and quality of the sounds. During auscultation of the lungs the patient usually sits upright and is instructed to breathe slowly and deeply through the mouth. The anterior and posterior surfaces of the thorax are auscultated from apex to base with comparisons made between the right and left sides. When the posterior chest is examined, the patient is asked to bring the shoulders forward so that a greater surface of lung can be auscultated.

Bronchodilator: A drug that dilates the airways in the lungs to improve breathing; works by relieving muscle contraction or buildup of mucus

Bronchospasm: The temporary narrowing of the airways in the lungs, either as a result of muscle contraction or inflammation; may be caused by asthma, infection, lung disease, or an allergic reaction

Dyspnea: A shortness of breath or a difficulty in breathing that may be caused by certain heart conditions, strenuous exercise, anxiety or a variety of pulmonary conditions

Emphysema: A chronic disease in which the alveoli become damaged; characterized by difficulty in breathing

Hemothorax: An accumulation of blood between the chest wall and the lungs

Hypercapnia: A $PaCO_2$ below 50 mmHg

Hyperoxia: A condition in which the tissue oxygenation is greater than what is required for cellular metabolism

Hyperoximia: A PaO_2 greater than 110 mmHg

Hyperventilation: A $PaCO_2$ less than 30 mmHg

Hypocapnia: A $PaCO_2$ above 30 mmHg

Hypoventilation: A $PaCO_2$ greater than 50 mmHg

Hypoxemia: A PaO_2 below 60 mmHg (in the patient below 60 years of age)

Intrathoracic: A location within the thoracic cavity

Metabolic Acidemia: An HCO_3 less than 20 mEq/liter

Metabolic Alkalosis: An HCO3 greater than 30 mEq/liter

Nasopharynx: The passageway connecting the back of the nose to the top of the throat

Normovolemic: having a normal volume of circulating fluid (plasma) in the body

Oxyhemoglobin: Hemoglobin that contains bound O2, a compound formed from hemoglobin on exposure to alveolar gas in lungs, with formation of a covalent bond with oxygen and without change of the charge of the ferrous state

Pneumothorax: A condition in which air enters the space between the chest wall and the lungs, causing chest pain and shortness of breath; may occur spontaneously or be the result of a disease or an accident

Polarographic: Pertaining to polarography

Pyothorax: thoracic empyema

Rales: An abnormal crackling or bubbling sound heard in the lungs during breathing

Respiratory Acidosis: A PaCO2 greater than 50 mmHg

Respiratory Alkalosis: A PaCO2 greater less than 30 mmHg

Tachycardia: A rapid heart rate (over 100 beats per minute)

Tracheotomy: Insertion of a tube through a surgical opening in the trachea to maintain an open airway

ICU crisis management update

Patient Safety in Surgery

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Abstract

Background:
Improving patient safety is an increasing priority for surgeons and hospitals since sentinel events can be catastrophic for patients, caregivers, and institutions. Patient safety initiatives aimed at creating a safe operating room (OR) culture are increasingly being adopted, but a reliable means of measuring their impact on front-line providers does not exist.

Methods:
We developed a surgery-specific safety questionnaire (SAQ) and administered it to 2769 eligible caregivers at 60 hospitals. Survey questions included the appropriateness of handling medical errors, knowledge of reporting systems, and perceptions of safety in the operating room. MANOVA and ANOVA were performed to compare safety results by hospital and by an individual's position in the OR using a composite score. Multilevel confirmatory factor analysis was performed to validate the structure of the scale at the operating room level of analysis.

Results:
The overall response rate was 77.1% (2135 of 2769), with a range of 57% to 100%. Factor analysis of the survey items demonstrated high face validity and internal consistency ($\alpha = 0.76$). The safety climate scale was robust and internally consistent overall and across positions. Scores varied widely by hospital [MANOVA omnibus F (59, 1910) = 3.85, $P < 0.001$], but not position [ANOVA F (4, 1910) = 1.64, $P = 0.16$], surgeon (mean = 73.91), technician (mean = 70.26), anesthesiologist (mean = 71.57), CRNA (mean = 71.03), and nurse (mean = 70.40). The percent of respondents reporting good safety climate in each hospital ranged from 16.3% to 100%.

Conclusions:
Safety climate in surgical departments can be validly measured and varies widely among hospitals, providing the opportunity to benchmark performance. Scores on the SAQ can serve to evaluate interventions to improve patient safety.

Hospitals are under increasing pressure to develop sound hospital systems to prevent sentinel events. The advancement of a culture promoting patient safety is a fundamental part of a systems approach to patient care and the administrative focus of many departments of surgery.[1] Recent attention to this topic stems from several high-profile medical errors and several Institute of Medicine reports which quantified the problem, created standardized definitions, and charged the healthcare community to develop improved hospital operating systems.[2,3] The promotion of patient safety has been further advanced by the recent malpractice crisis in surgery and the demonstrated vulnerability and devastation hospitals face after public exposure of a sentinel event. Compared with other hospital settings, errors in the operating room can be particularly catastrophic and, in some cases, can result in high-profile consequences for a surgeon and an institution. Wrong-site/wrong-procedure surgeries, retained sponges, unchecked blood transfusions, mismatched organ transplants, and overlooked allergies are all examples of potentially catastrophic events which, in certain circumstances, can be prevented by improved communication and safer hospital systems. In one study of all root cause analyses submitted to the Joint Commission on Accreditation of Healthcare Organizations (JCAHO), communication was identified as the most common root cause of sentinel events wrong-site surgeries.[4] As a result, creating a culture of safety is a high priority for surgeons and hospitals.

Several interventions to improve patient safety in surgery have been introduced, including additional checks to confirm procedures and new policies to govern the operating room. In addition, many hospitals are investing in safety training programs for their staff in an effort to improve the culture of safety in the operating rooms. Yet, while there are many new safety initiatives, there are few tools available to measure the actual effect of interventions on outcomes. This is a critical problem in validating patient safety improvement efforts. Furthermore, collecting data on medical errors in surgery is difficult because near misses are often unreported and sentinel events can be rare. Using a valid and reliable measurement instrument, culture data can serve as a benchmark for hospitals to gauge their performance in advancing the patient safety agenda.

Applying a fundamental axiom of business management, we maintain that accurate and scientific feedback from front-line personnel is a critical component of any successful intervention. Indeed, attitudes about culture among workers have been associated with error reduction behaviors in aviation,[5] and with patient outcomes in intensive care units.[6–8] Based on this demonstrated association in the literature and our own clinical experience, we propose that perceptions of *how safe a workplace is,* as recognized by front-line providers, is a reliable and valid surrogate of adverse events. Indeed, it is perhaps the only surrogate we have in measuring safety risk. An “unsafe” operating room culture, as assessed by front-line providers, can in fact be an important risk factor for the occurrence of a sentinel event.

Recognizing the potential association between culture and outcomes, the JCAHO is proposing a requirement that all hospitals measure their culture beginning in 2007 (www.jcaho.org). Hospitals are encouraged to start measuring culture in the year prior to the new requirement. While there are many assessment surveys for quality of life and other aspects of well-being, there are no reliable measurement tools for culture that have been widely adopted in the surgical setting. The primary aims of this study were: 1) to test the reliability of a safety climate scale to assess group-level consensus or “climate” in the surgical setting, and 2) to provide useful benchmarking information on safety culture from 60 U.S. hospitals. Secondary objectives of this study were to examine differences in safety culture as a function of hospital and position (surgeon, anesthesiologist, certified registered nurse anesthetist [CRNA], OR nurse, and technician).

METHODS

The surgical survey instrument that we developed and used in the current study, the Safety Attitudes Questionnaire[9,10] was adapted from the Flight Management Attitudes Questionnaire (FMAQ)[11] and its predecessor, the Cockpit Management Attitudes Questionnaire (CMAQ).[12] The CMAQ is reliable, sensitive to change,[13] and elicited attitudes shown to predict performance.[5,14] We improved content validity of the SAQ by reviewing the literature on patient safety in the OR, asking OR healthcare provider types to review the survey, and by conducting focus groups.

The SAQ measures 6 domains: teamwork climate, safety climate, job satisfaction, perceptions of management, stress recognition, and working conditions. Seven of the 30 SAQ scale items map onto the safety climate domain. Safety climate assesses the perception of a strong proactive organizational commitment to safety, and our group has found that the elicited attitudes are associated with patient length of stay and error rates in the ICU.[15] In this manuscript, we report the results of the safety climate domain in OR caregivers.

The SAQ (OR version) was administered to all OR caregivers in a Catholic health system comprised of 60 hospitals in 16 states in July and August of 2004. No provider type was excluded and OR caregivers included surgeons, anesthesiologists, CRNAs, OR nurses, and surgical technicians. Random sampling was not used due to small sample sizes in caregiver positions with a hospital, which would threaten the representativeness of the data. Instead, we sought as high a response rate as possible within each caregiver type within each hospital. Surveys were administered during preexisting departmental and staff meetings, together with a pencil and a sealable return envelope to maintain confidentiality. Individuals not captured in preexisting meetings were hand delivered a survey, pencil, and return envelope. No personal identifying information was tracked beyond job position and hospital.

We used multilevel confirmatory factor analysis[16] and reliability analyses to evaluate the reliability and preliminary validity of the 7-item safety climate scale. A basic criterion required to adequately assess culture or climate constructs is that individual perceptions show high agreement *within* units and high variance *between* units.[17] To examine the level at which perceptions of safety are shared, we first examined 2 units of analysis: professional culture (ie, by OR provider) and hospital culture (ie, by surgical unit). We tested for differences between OR providers and differences between hospitals with respect to each item using MANOVA, and globally using ANOVA. Then, to evaluate the extent to which perceptions of safety are shared within units and vary between units, we examined 2 versions of the intraclass correlation (ICC1 and ICC2) as well as the $r_{wg(j)}$ interrater agreement statistic.[18] Safety climate scale scores were computed by taking the average of the 7 items. One item was reverse scored with opposite wording valence to confirm accuracy. All statistical analyses were performed using SPSS version 12.0 (Chicago, IL) and MPLUS version 2.01.

RESULTS

There were 2769 eligible subjects (222 surgeons, 1058 OR nurses, 564 surgical technicians, 170 anesthesiologists, and 121 CRNAs) from the 60 hospitals. The overall response rate was 77.1% (2135 of 2769), with a range across hospitals of 57% to 100%. Table 1 shows respondent demographics and response rates.

TABLE 1. Respondent Demographics

TABLE 1. Respondent Demographics

Position	Response Rate (Returned/Administered)	Age (Yr) (Mean ± SD)	% Female (No.)	Years of Experience in Position (Mean ± SD)	Years Working at Current Hospital (Mean ± SD)
Surgeon	73% (222/305)	48.3 ± 9.92	8.6% (19)	17.4 (9.41)	12.3 ± 9.20
Surgical technician	78% (564/728)	37.8 ± 11.58	73.7% (417)	11.2 (11.17)	7.9 ± 9.45
Anesthesiologist	77% (170/220)	45.8 ± 9.31	12.7% (21)	15.8 (8.18)	10.6 ± 8.60
CRNA	67% (121/181)	44.6 ± 10.71	50.0% (63)	14.7 (12.32)	9.5 ± 9.35
OR nurse	79% (1058/1335)	43.3 ± 10.85	89.0% (942)	13.9 (10.04)	10.7 ± 8.69
Total	77% (2135/2769)	42.6 ± 11.3	68.5% (1462)	13.7 (10.47)	10.0 ± 9.08

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Safety climate varied widely by hospital, but not position (Figs. 1, 2). MANOVA of the 7 items yielded 2 significant omnibus *F* results. An omnibus F for OR healthcare provider type of F (28, 6059) = 2.19, $P < 0.001$. Further analyses with Bonferroni adjustment specifically revealed that OR nurses were less positive about one item “I would feel safe being treated here as a patient” than surgeons and anesthesiologists. In other words, 6 of the 7 items did *not* differ significantly by OR provider. An omnibus F for Hospital of F (413, 11700) = 1.76, $P < 0.001$, indicating that respondents perceive safety climate issues differently as a function of the hospital in which they work.

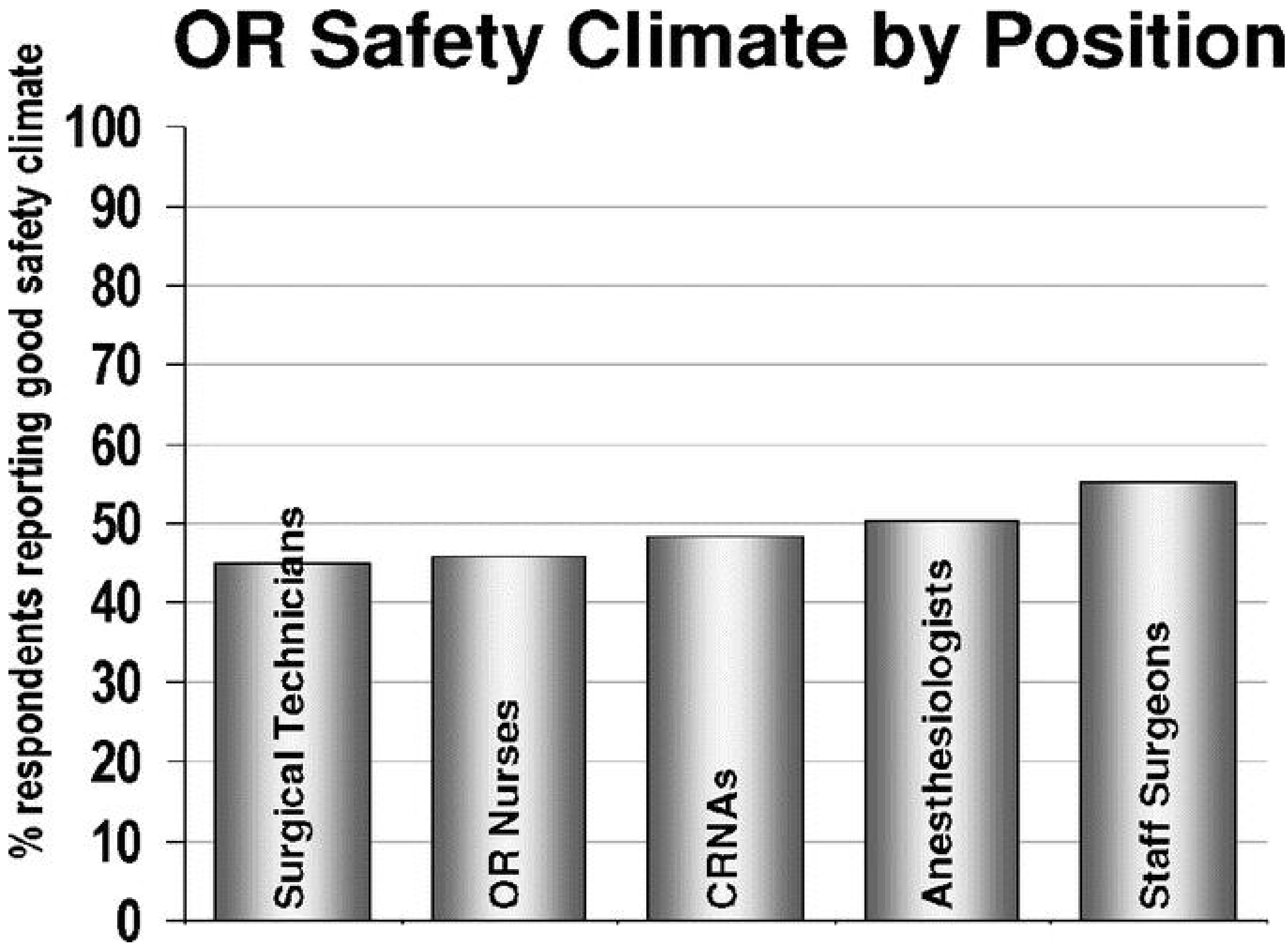

FIGURE 1. Safety climate by position.

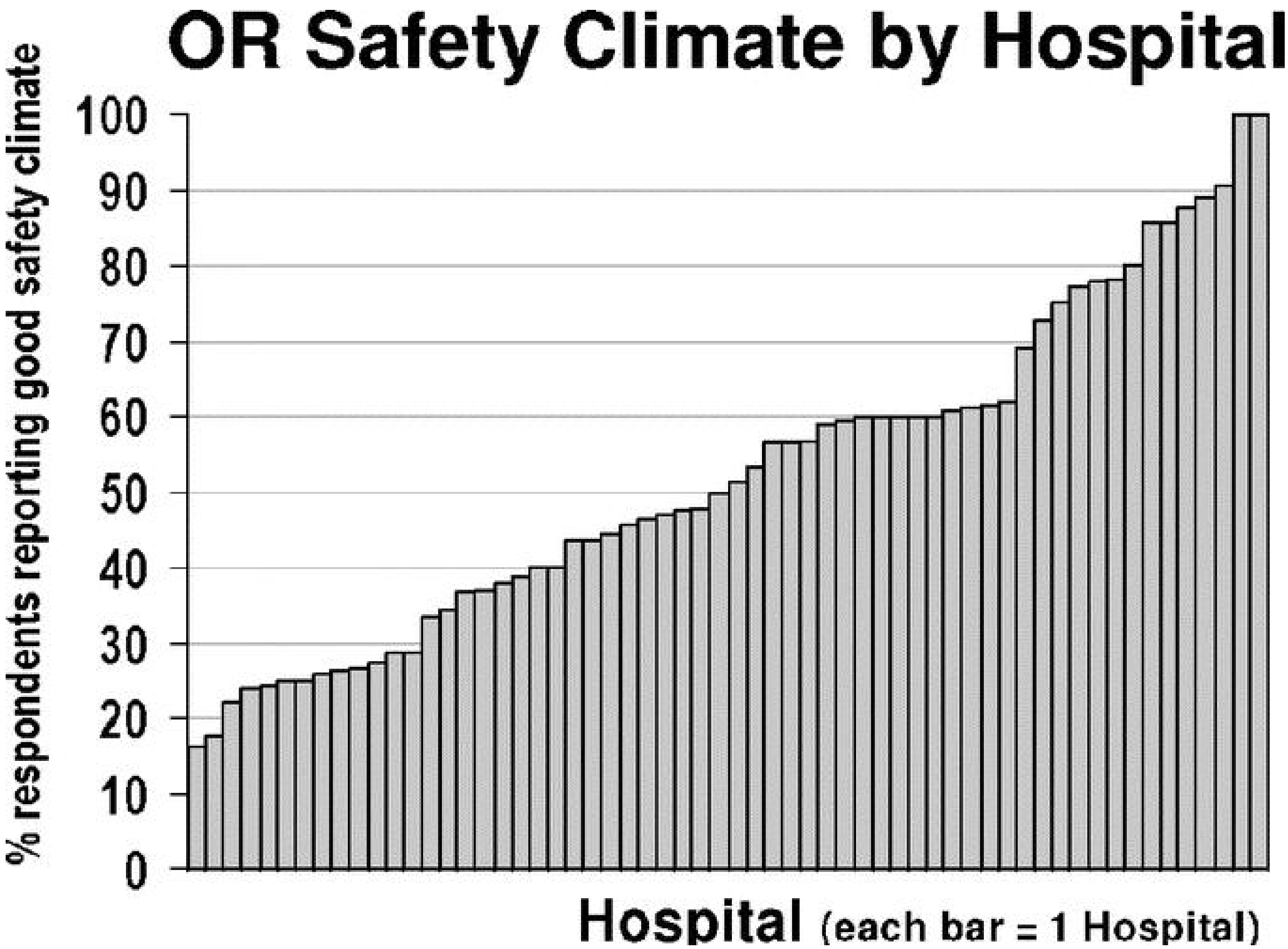

FIGURE 2. Safety climate by hospital (each bar represents one hospital).

For the scale score analyses, ANOVA demonstrated no significant differences in safety climate scale scores between OR healthcare provider types, F (4, 1910) = 1.64, $P = 0.163$: surgeon (mean, 73.91), surgical technician (mean, 70.26), anesthesiologist (mean, 71.57), CRNA (mean, 71.03), and OR nurses (mean, 70.40). However, there were significant differences between hospitals (F (59, 1910) = 3.85, $P < 0.001$). Table 2 demonstrates the percent agreement (agree slightly and agree strongly) by position and by hospital. The percent of respondents reporting good safety climate in each hospital ranged from 16.3% to 100%.

DISCUSSION

The question "How do we know we are safer?" poses a challenge to many surgeons and institutions. The framework of understanding safety is Donabedian's model of categorizing measures as measures of structure, process, or outcome.[19] Structure is how we organize care, process is what we do, and outcomes are what we achieve (Fig. 3). The context of this system is the local culture, which affects each component in that it is how front-line personnel understand safety. In an attempt to analyze safety, many institutions and collaborative organizations focus on measures of structure (eg, presence of policies or committees). They may also measure processes (eg, how often evidence-based interventions are performed) and outcomes (eg, how often patient's are harmed). While these data can be useful in guiding system changes, measuring structure, processes, or outcomes in isolation may be misleading and ignores how engaged front-line personnel are in delivering safe patient care (Fig. 4). Creating a culture of safety is a paramount priority for many departments of surgery. While this goal is held high, measuring culture is rarely performed since the field of culture measurement in the healthcare setting has traditionally lacked good scientific methods.

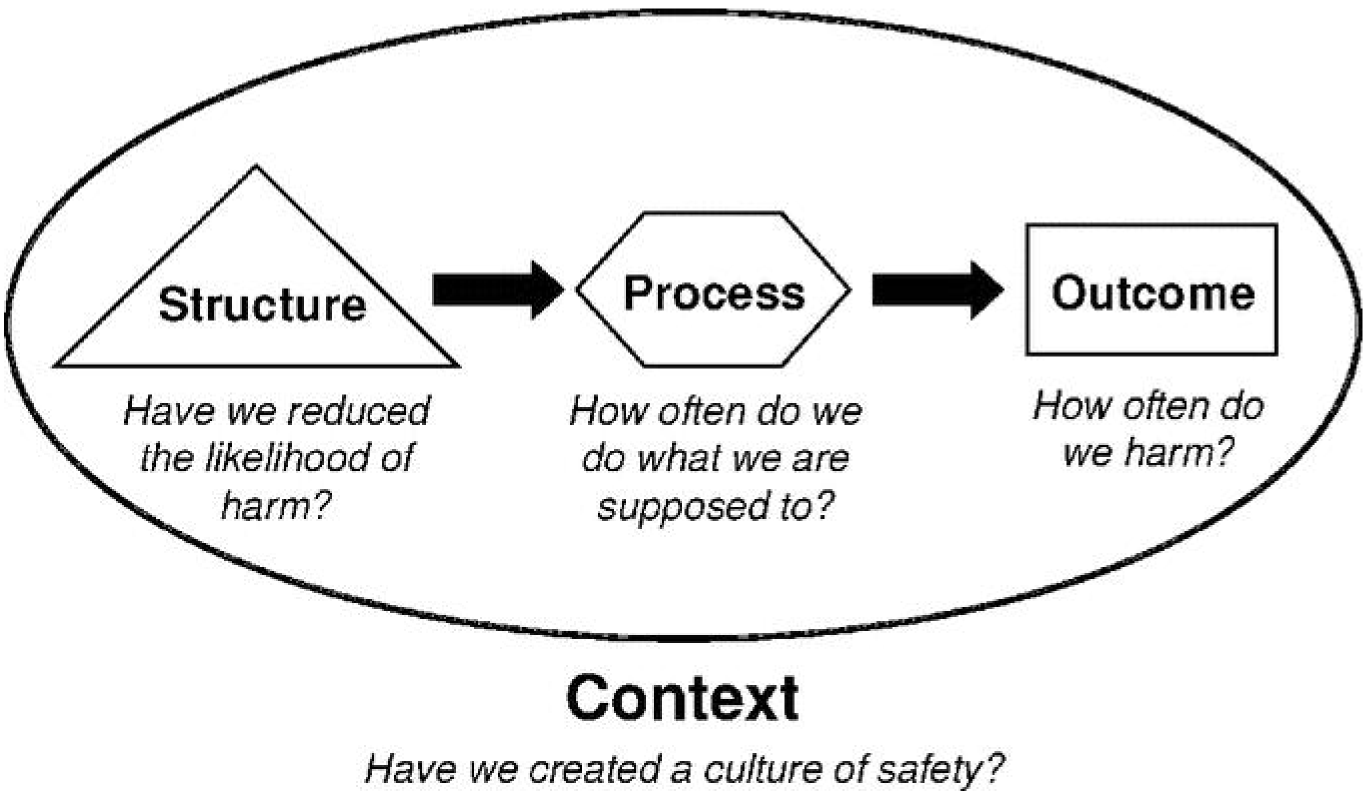

FIGURE 3. Donabedian model for measuring quality. Type of measures: structure, process, outcome, or culture.

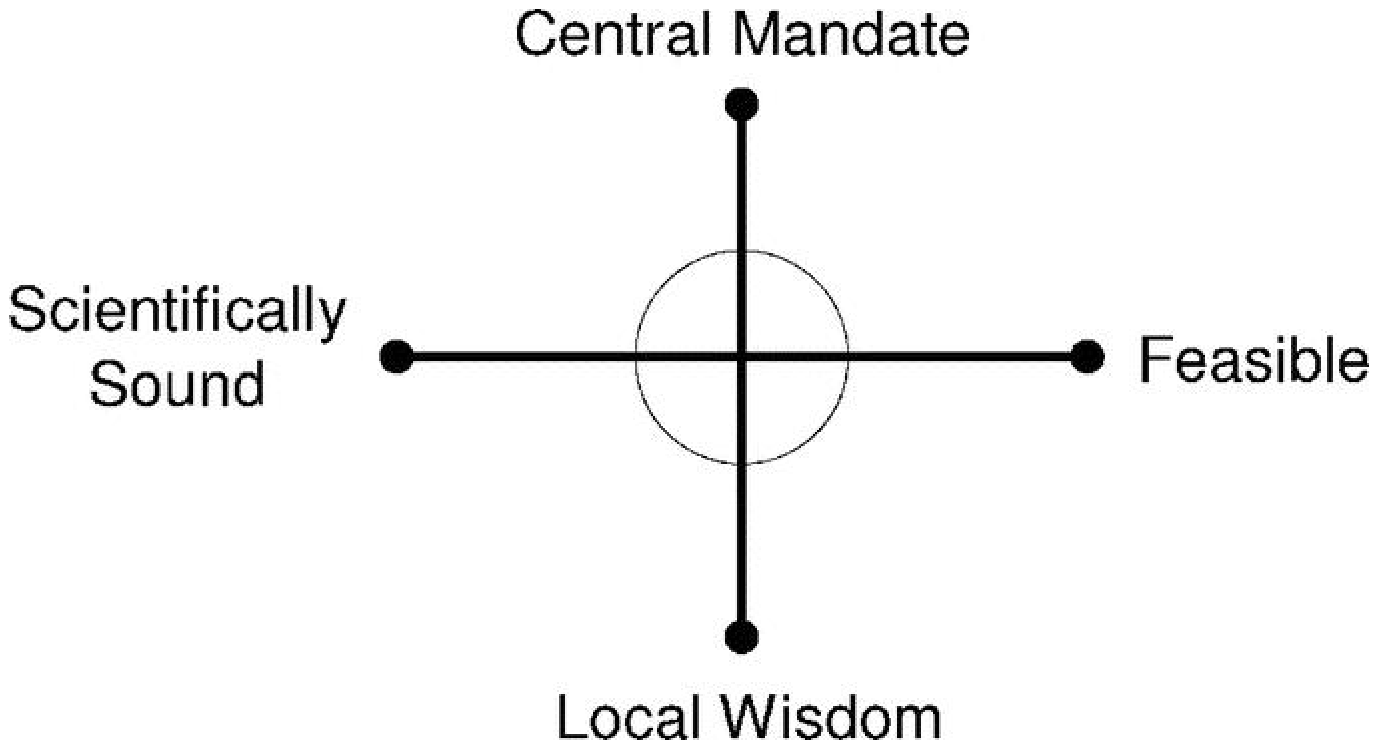

FIGURE 4. Finding the sweet spot: Issues in improving patient safety.

The recent identification of safety culture as an important factor of a hospital system by JCAHO has spurred many hospitals to find a scientifically sound method to measure culture. We propose that OR safety culture can be measured using the safety climate scale of the SAQ. This psychometrically valid assessment provides benchmarks for departments of surgery and hospitals seeking to compare their safety climate to national means. In addition, it can serve as a baseline measure for evaluating any safety intervention. Unlike perceptions of teamwork climate, which differ as a function of role in the OR,[10] perceptions of safety climate are relatively consistent across OR providers in a given hospital. However, the marked variation in hospitals' safety climate scores, and the evidence that safety climate is sensitive to interventions,[15] suggests that existing strategies to promote patient safety at some centers may be effective. Identification and dissemination of these best practices could potentially benefit the surgical community at large. In effect, the survey elicits the input of the front-line personnel in surgery, recognizing that they have an important operational perspective on patient safety.

Limitations

The results reported here represent findings from 60 hospitals in one system. Organizations and researchers wishing to use this information for benchmarking purposes should be aware of 2 limitations. Although originally designed to be a baseline assessment, many of the hospitals had already implemented specific interventions aimed at improving patient safety. Consequently, even though the results identify significant opportunities for improvement, the overall distribution across the system may be higher than expected for a true baseline assessment. Another potential limitation relates to the remarkable response rates obtained (Table 1) that may be difficult for other organizations to obtain without methodologic rigor and support from senior leadership.

Teamwork

A rapidly growing industry of teamwork training programs has emerged to meet the growing demand to make operating room staff more aware of teamwork and communication as a means to improve patient safety. However, current teamwork training initiatives have not effected long-term attitudinal or behavioral changes. While these programs can be helpful, we have found that they are most effective in a peer-to-peer format, ie, surgeons teaching surgeons based on their experience and existing data (Fig. 4). Changing a culture is difficult and is best accomplished through the use of "physician-champions" who serve as local role models and drivers of change. Empowering well-respected surgeons to promote principles of teamwork and communication can be the most effective means to advocate safe operating room practices.

OR-ICU Briefings and Debriefings

One strategy to improve patient safety in surgery adopted at the Johns Hopkins Hospital is the use of OR-ICU briefings and debriefings. These discussions, initiated and led by the surgeon, are intended to prevent and mitigate adverse events by promoting communication through improved teamwork. Specifically, they encourage any team member to speak up if they perceive a problem that could result in patient harm. The briefing consists of introductions by first name and role of each OR team member, a surgical time-out (or pause), and discussion of expectations for the operative plan, paying special attention to potential problems that could be encountered. We also conduct debriefings at the end of the case to note lessons learned for future patients and procedures. Preliminary evidence suggests that preoperative OR briefings are associated with an improved safety culture, reductions in wrong-site/wrong procedure surgeries, early reporting of equipment issues, and reduced operational costs.[20] Although briefings and debriefings are not end-all solutions to the problem of errors or inefficiencies in the operating room, they help to minimize errors by allowing personnel to discuss potential problems before they lead to a "near miss" or actual harm. Ultimately, what is needed is a combination of innovative surgical systems, a sustained focus on patient safety, improved communication, and excellent providers to reduce the risk of errors and enable surgical outcomes to achieve a six-sigma state.

Implications

Measurement is the foundation of quality improvement. In industry, tracking predictors of performance is vital to implementing new business strategies. In the hospital, culture scores may be the most sensitive tool available to measure safety risk in an operating room. Front-line providers have unique insights into the reliability and quality of any system. Indeed, executive interaction with front-line personnel is heralded as a marker of good leadership. Based on our findings, we submit that the SAQ may represent one approach for leaders in surgery to better understand the operating room, and, specifically, areas within a surgical department where the culture could be improved.

DISCUSSIONS

Dr. John B. Hanks (Charlottesville, Virginia): Dr. Makary and Dr. Freischlag and their group are to be congratulated on a nice analysis of a problem that is receiving increasing attention, a systems analysis of quality of patient care. They stress the important points that attitudes about the patient care setting, work environment and, by inference, job satisfaction, all play an important part in understanding the essentials of improved patient care.

This is an extremely difficult area to get past old habits and perceptions. To be really intelligent about this area, we need quantitative data and tools, and this group has done an extremely well-defined approach. At UVA, we have used the time-out process specifically, and, given our experience with our recent JACHO review, this was received quite positively. So I suggest to membership that more than just a few of these things are very relevant.

I have a couple of questions for the authors.

As I understand the manuscript, the data are based on one mailing of the questionnaire. Do they have any experience with how the data might change on a second or third mailing? Would there be a Hawthorne effect? Do they feel that the experience of just going through the questionnaire itself might positively or perhaps negatively affect the answers later about a commitment to quality patient care?

As I interpret their questions, I see that there are really two types that are very interesting. One set of questions would relate to job satisfaction. The second obviously deals with issues of the quality, of which the most important question is, would I feel safe being operated on in this hospital?

Did the authors look at a stratification of job satisfaction and compare those levels against perceptions about quality of care? Neophytes in this area, such as myself, always worry about whether or not the less satisfied members of the team have colored their answers about quality of care.

Dr. Martin A. Makary (Baltimore, Maryland): Certainly the Hawthorne effect was present. It is intrinsic to all surveys. It is essentially a bias that participants have to overrepresent how well they are doing because they know they are being evaluated. In fact, I think that speaks even more to your excellent point that this survey needs to be given in sequence so that changes in culture are seen as sustainable.

Training events and safety programs which are measured by instant questionnaires at the conclusion of the program or the training session are, in my opinion, meaningless and not worth our time. Culture change has to be significant, especially since changes in culture appear to be related to the outcome measures that we have looked at as defined by AHRQ, namely, rates of surgical site infections, rates of postoperative hemorrhage and hematoma requiring a second operation, and rates of pulmonary embolism and DVT, as you saw on the slide.

The study design, of course, is not perfect. But it is feasible. And in balancing what is scientifically sound versus what is feasible, we think that a simple set of questions, namely, asking personnel whether or not they feel comfortable being operated upon in the operating rooms in which they work, is probably one of the best ways to not only fulfill the JCAHO requirement but provide a cheap and accurate means of risk assessment, which is internal and is developed by surgeons. This is a question that we came up with as a group of surgeons in conjunction with Brian Sexton, who is a social psychologist who actually worked for Continental Airlines looking at the same question. So it is minimally invasive.

I appreciate your comment, Dr. Hanks, that culture is very fluid. In fact, I think that is why all these hospitals are in fact interested in measuring their culture on an annual basis.

Job satisfaction has actually been an interesting domain. We added questions using the Moslatch scale. Dr. Moslatch was a social psychologist at the University of Michigan whose career was based on the science of burnout. And we added questions so that we could derive an index score based on her understanding of burnout from measuring it in numerous disciplines, not just medicine. And in fact, there was an association between job satisfaction burnout and safety climate, although when adjusted for job satisfaction, burnout, there were still significant differences in safety climate. So your point, I think, is a valid one. And whether or not there is a cause and effect, I think perhaps is best determined locally, or future research may show the Association.

The purpose of this study was really to provide a simple and scientifically valid and reliable tool to measure culture in some form. The alternative, of course, from the psychometric standpoint, would be to have independent observers come into the operating room and watch behavior. In fact, we do have a study at Kaiser looking at 350 operations where two social psychologists are observing how often surgeons engage in a conflict and how those conflicts are resolved. And they look at specific features of team dynamics all the way down to whether or not we know the names of the people we work with. This simple and well-validated questionnaire, I would submit is a much cheaper and better approach to measuring culture, and in fact will fulfill the JACHO requirement that is pending for 2007.

Like NSQIP, this is essentially a response that we, I think, as surgeons have an opportunity to develop as a group without collaborative organizations and groups of non-surgeons telling us how we need to do this. And I think it is perhaps one effective way to measure culture.

Dr. Thomas R. Russell (Chicago, Illinois): I can tell you that, at the American College of Surgeons, we are taking very seriously the issue of not only safety, which focuses on adverse events or mistakes that happen in hospitals, but also quality improvement, which attempts to set up processes of care in our hospitals so that these mistakes never happen.

You alluded to the Joint Commission on Accreditation of Healthcare Organizations (JCAHO). The JCAHO is really attempting to transform itself into an organization that is not just an accrediting body or a disciplinary body, but rather an institution that is coming into our hospitals with concrete efforts to improve processes of care. The universal protocol that dealt with correct site and patient surgery was established a few years ago with the American College of Surgeons and other interested stakeholders. You are going to see an increased number of core safety measures in quality improvement that will be emanating from the JCAHO.

I commend Johns Hopkins for doing this study. You are leading an effort in safety with appropriate alignment between the medical staff and the administration of the hospital. This is absolutely going to be key to have a lead organization such as Johns Hopkins take charge in trying to bring a strong culture of safety into the fabric of our hospitals.

I would like to ask the question to whether you are extending this type of survey into other components at Johns Hopkins? Are you taking it from the operating rooms and dealing with other aspects of health care within the hospital?

Dr. Martin A. Makary (Baltimore, Maryland): We have modified the SAQ to the intensive care unit. That is the first domain that we have expanded to. We have also now a version for the emergency department and labor and delivery area and in fact, every clinical area, so that an institution can measure their culture hospital-wide.

I think it is important, as you mentioned, that the local wisdom be accounted for in assessing culture. There is a constant tension and sort of quality measurement universe of central mandates versus local wisdom, and we have been propagating very strongly for local wisdom to be a part of every metric that is being developed within surgery.

Essentially, this is consistent with any management strategy, that is to account for the perspectives of front-line workers in measuring the level of safety or quality in any process. So I very much appreciate your comments.

Dr. Joseph B. Cofer (Chattanooga, Tennessee): I have been doing something a little bit different at my place. For about 6 months now, I have been using a “pre-flight” checklist, based on my time in the Navy, where before surgery I have a “pre-flight” brief in the holding area between anesthesia, surgery, and the nurses. There is a form that is filled out. We do the case, we have a time out. Then afterwards we have a 360 evaluation wherein nurse, anesthesia, and surgery all evaluate each other.

I have found that for me it has worked great, especially in getting preference cards read, blood always ready, antibiotics always given, etc. The nurses really love it. They really think it is great. But I cannot get anesthesia on board. It is almost like they are offended that you would want to meet with them in the holding area and discuss anesthesia or discuss how the case is going to be done or discuss vascular access.

Have you had that problem? The nurses love it. The patients seem to like it. They see what is going on. They are awake, you are at the foot.

Dr. Martin A. Makary (Baltimore, Maryland): We have had every problem you can conceive of, including that problem. Certainly, changing a culture takes time. And I applaud you for your effort. Certainly, the briefing as we outlined is not perfect, and that will be the first or second of many renditions that will develop through the years as we figure out what works best both for surgery and within our own hospitals.

I have also noticed that the patients love it when we discuss their concerns, if they have a concern about their airway or they want to make sure they are going to receive local anesthetic or if they are not going to receive blood products or that they have a certain allergy. When they hear the discussion going on or know that it is going to go on before their operation, they are almost sometimes shocked that this hasn't been going on for years as a routine sort of conversation among groups. So I think what you are doing is excellent. In fact, I think we will all learn together.

In the distribution of safety climates that we saw among the 60 hospitals, what we have done is take individuals like yourself that have had some success and some frustrations and share those experiences with the hospitals that have a low safety climate. And from dialogues like this perhaps we can all learn from each other.

Dr. Aaron S. Fink (Atlanta, Georgia): In the VA, many of these things are mandated at a national level. We had the good fortune of being visited by the National Patient Safety Center, which has avidly promoted both actions, particularly the preoperative briefings that you have instituted. We had a lot of trouble with "buy-in" for preoperative briefings, as many considered this to be an exaggerated time-out; thus, we focused our efforts on developing a debriefing, as you have.

We found that the debriefing has been better accepted by many of our staff in the operating room. In addition, it has provided an opportunity to focus discussion between the attendings and the residents regarding to the four measures of the SCIP program being recommended by CMS. Thus, we have now formalized a discussion about whether antibiotics will be used in a prophylactic manner and, if so, which ones and when they will be discontinued; thromboembolic prophylaxis if the patient has a cardiac situation; whether beta blockers have been started and will be continued; and obviously for patients on the ventilator, what measures will be instituted in addressing ventilator-associated pneumonia. We have found this approach to be very helpful.

Dr. C. Daniel Smith (Atlanta, Georgia): I don't want to sound like a skeptic because I think this time-out would be incredibly valuable. However, in the course of my operative day, I am the only consistent member of my team from beginning to end. There will be no less than 10 team changes: anesthesia, nurses. Maybe you could give some practical recommendations on how you deal with this team migration and change that takes place. Actually, by the end of a case, my team has changed, so the debrief at the end won't consist of the same team members we started with. How do you deal with this during the course of an operative day in a busy practice where you will have residents not even finish the day with me in the operating room?

Dr. Martin A. Makary (Baltimore, Maryland): That is an excellent point. We as attending surgeons often find ourselves the only non-shift workers in the operating room sometimes. And it is a difficult problem, to be very frank. We have tried to do some things such as make it a rule that nurses need to change their name on the board of the operating room when they come in, and we are trying to minimize the number of silent switch-outs, so that people just feel encouraged to say, "I'm here now taking over for so-and-so, and what's going on?"

The idea is to encourage approachability and to get people to speak up if they see that the blood type is not right for the heart that is being transplanted or some major catastrophe. If they feel that antibiotics should have been administered to a patient but somebody may have forgotten, we want to encourage people to speak up and say something. Certainly, a briefing, a checklist covering antibiotics, DVT prophylaxis, is not going to cover everything and it is not perfect. But it is some discussion that we can have with our colleagues.

This field of patient safety has been really plagued with bad science for many years. And the only science that we have is really from industry and from aviation. I have found that surgeons sometimes resent being compared to pilots because things are not so well measured in the operating room. Perhaps with anesthesia, that may be different. Anesthesiologists feel that giving anesthesia is very much like flying a plane, that they use a lot of the same lingo. But in surgery, there are many more variables that are difficult to measure.

The idea of talking about the lack of antibiotic prophylaxis, the high rates of DVTs, comparing your performance within a collaborative, and using data such as data from small studies of briefings may be one attempt to apply some science to a field that really has had very little science to an audience which really speaks the language of science. And we have found that in achieving buy-in, which is a very difficult problem, identifying physician champions can be the best means of doing that.

This concludes the 2007 UPDATE MATERIAL for this ICU Crisis Management Course.

POST TEST

1. SIMV is usually implemented for which of the following reasons?

   A. To reduce the mean intrathoracic pressures
   B. To control the patient's breathing
   C. To wean from mechanical ventilation
   D. To improve the V/Q ratio
   E. To force the patient to breathe spontaneously

2. Which of the following is a good guideline for determining when PEEP should be initiated in the patient who is already receiving mechanical ventilation in an assist-control mode?

   A. A P_aO_2 of 70 mmHg or less on 40% oxygen
   B. A P_aO_2 of 50 mmHg or less on 40% oxygen
   C. A P_aO_2 of 70 mmHg or less on 50% oxygen
   D. A P_aO_2 of 50 mmHg or less on 50% oxygen
   E. A P_aO_2 of 40 mmHg or less on 50% oxygen

3. Which of the following can be complications associated with PEEP?

   I. Atelectasis
   II. Pneumothorax
   III. Reduced cardiac output
   IV. Decreased V/Q

   A. II
   B. I and IV
   C. I, II and III
   D. I, II and IV
   E. I, II, III and IV

4. Which of the following represents the order that should be taken when discounting PEEP?

   A. First reduce the minute ventilation to less that 10 lpm, then reduce the PEEP to 0 in increments of 5, and finally reduce the F_IO_2 to 40%.
   B. First reduce the minute ventilation to less than 10 lpm, then reduce the F_IO_2 to 40%, and finally reduce the PEEP to 0 in increments of 5.
   C. First reduce the F_IO_2 to 40% and then reduce the PEEP to 0 in increments of 5.
   D. First reduce the F_IO_2 to 40%, then reduce the minute ventilation to less than 10 lpm, and finally reduce the PEEP to 0 in increments of 5.
   E. First reduce the PEEP to 0 in increments of 5 and then reduce the F_IO_2 to 40%.

5. Which of the following is an appropriate application of pressure support ventilation (PSV)?

I. To improve the FRC thereby improving the oxygenation
II. To decrease the resistance to breathing caused by the ET tube
III. To improve the compliance during spontaneous breathing
IV. To decrease the work of breathing during spontaneous breathing

A. I and II
B. II and III
C. II and IV
D. I, II and IV
E. II, III and IV

6. Which of the following describe the characteristics of vesicular breath sounds?

A. Can be heard predominately in the upper lobes
B. The inspiratory phase is shorter than the expiratory phase
C. The sounds have a tubular quality
D. There are no sounds present during the expiratory phase
E. There is no pause between inspiration and expiration

7. Which of the following diseases is/are usually associated with increased amounts of sputum production?

I. Bronchiectasis
II. Chronic bronchitis
III. Cystic fibrosis
IV. Lung cancer

A. I and II
B. II and III
C. I, II and III
D. II, III and IV
E. I, II, III and IV

8. What is the best way of determining whether a patient is dyspneic?

A. Arterial blood gases
B. Arterial blood gases and chest x-ray
C. Bedside spirometry
D. Chest auscultation
E. Patient history

9. Which of the following is/are advantages of an arterial line?

I. Permits frequent blood gas analysis
II. Reduces the incidence of infection

III Provides a means for monitoring the arterial pressures
IV Provides a means for obtaining a pulmonary wedge pressure

A. I only
B. I and II
C. I and III
D. I, II and III
E. I, II, III and IV

10. The normal CVP is about:

A. -5 to +5 cmH_2O
B. -5 to 0 cmH_2O
C. 0 to +5 cmH_2O
D. +5 to +15 cmH_2O
E. +10 to +25 cmH_2O

11. A reduced CVP may indicate which of the following phenomena?

I. Reduced cardiac output
II. Fluid overload
III. Poor tissue oxygenation
IV. Hypovolemia

A. I only
B. IV only
C. I and II
D. I and III
E. I and IV

12. Which of the following can be obtained from a Swan-Ganz line?

I. Arterial blood gases
II. Pulmonary wedge pressures
III. Cardiac output determinations
IV. Pulmonary artery pressure readings

A. II and IV
B. I, II and III
C. I, III and IV
D. I, II and IV
E. II, III and IV

13. An increase in the pulmonary wedge pressure indicates which of the following?

A. Pulmonary artery hypotension
B. Reduced blood volume returning to the heart
C. Systemic hypertension

D. Increased left atrial pressure
E. Increased cardiac output

14. Hypoxia can be defined as follows:

A. A P_aO_2 less than 100 mmHg
B. A P_aO_2 less than 50 mmHg
C. A subjective sensation of air hunger
D. Failure of oxygen to cross the alveolar-capillary membrane
E. Inadequate tissue oxygenation

15. What parameters are sufficient for determining the adequacy of tissue oxygenation?

I. The P_aO_2
II. The S_aO_2
III. The C_aO_2
IV. The cardiac output

A. I only
B. II only
C. I and II
D. I and IV
E. III and IV

16. The adequacy of alveolar ventilation is best assess by which of the following assessment procedures?

A. The exhaled minute ventilation
B. The P_aO_2
C. The P_aCO_2
D. Observation of the respiratory pattern
E. The patient’s subjective sensation

17. On a chest x-ray examination, an elevated diaphragm could be the result of which of the following?

    A. Pleural effusion
    B. COPD
    C. Tension pnuemothorax
    D. Asthma
    E. Atelectasis

18. A deviation of the trachea to the right side of the chest could be the result of which of the following?

    A. Right pleural effusion
    B. Right tension pneumothorax
    C. Ball-type obstruction in the right mainstem bronchus
    D. Atelectasis on the right side
    E. Large bullae in the right lung

19. A disease associated with destruction of the pulmonary septal walls and connective tissue beyond the terminal bronchioles is:

    A. Cystic fibrosis
    B. Asthma
    C. Chronic bronchitis
    D. Bronchiectasis
    E. Emphysema

20. What are the typical blood gases for COPD patient in mild distress?

    A. Uncompensated respiratory acidosis and hypoxemia on room air.
    B. Compensated respiratory acidosis and hypoxemia on room air
    C. Uncompensated respiratory acidosis and normal oxygenation on room air
    D. Respiratory alkalosis and normal oxygenation on room air
    E. Normal acid-base and hypoxemia on room air

21. Wheezing may respond to which of the following medications?
    I. Albuterol
    II. Aminophylline
    III. Morphine
    IV. Lasix

    A. I only
    B. I and II
    C. I, II and III
    D. I, II and IV
    E. I, II, III and IV

22. The cardinal sign of asthma is:

A. Dyspnea
B. Decreased FRC
C. Anxiety
D. Tachycardia
E. Wheezing

23. The chest x-ray of the asthmatic patient in acute exacerbation will have the following characteristics:

I. Elevated ribs
II. Elevated diaphragm
III. Decreased translucency

A. I only
B. II only
C. I and II
D. II and III
E. I, II and III

24. Which of the following arterial blood gas values are representative of the asthmatic patient in mild exacerbation on room air?

A. pH 7.30, P_aCO_2 48 mmHg, P_aO_2 85 mmHg
B. pH 7.42, P_aCO_2 48 mmHg, P_aO_2 85 mmHg
C. pH 7.42, P_aCO_2 48 mmHg, P_aO_2 60 mmHg
D. pH 7.45, P_aCO_2 35 mmHg, P_aO_2 60 mmHg
E. pH 7.30, P_aCO_2 48 mmHg, P_aO_2 60 mmHg

25. ARDS can be associated with a variety of injuries. From the time of the injury, when does ARDS usually begin to become clinically apparent?

A. Within 2-4 hours
B. Within 4-12 hours
C. Within 12-48 hours
D. Within 36-48 hours
E. Within 48-72 hours

26. Which of the following is a pathophysiological factor in ARDS?

A. Increased compliance of the lung
B. Increased airway resistance
C. Increased permeability of the alveolar-capillary membrane
D. Decreased pulmonary vascular resistance
E. Increased histamine production

27. What is the most decisive laboratory or clinical finding in ARDS?

A. Interstitial and alveolar fluid accumulation on the chest x-ray

B. Hypoventilation while breathing spontaneously
C. Diminished breath sounds bilaterally
D. Hypoxemia despite high concentrations of oxygen
E. Decreased cardiac output

28. Which of the following is a cardinal sign or clinical symptom of a flail chest resulting from blunt chest trauma?

A. Dyspnea
B. Tachypnea
C. Hypoventilation
D. Paradoxical motions
E. Use of accessory muscles

29. What is the primary treatment for a flail chest?

A. Close observation and supplemental oxygen
B. IPPB therapy and sedation
C. Strapping the chest and sedation
D. IPPB therapy and supplemental oxygen
E. Intubation and mechanical ventilation

30. Which of the following statements characterizes myasthenia gravis?

A. Usually occurs in children
B. A chronic respiratory disease with periodic relapses
C. An acute respiratory disease that is easily treated pharmacologically
D. Affects only the respiratory muscles
E. Restrictive lung dysfunction during acute exacerbation

31. Which of the following is characteristic of Guillain-Barré?

A. Paralysis usually begins in the upper body and descends
B. Usually preceded by an upper respiratory tract infection
C. Large amounts of thicker, mucoid secretions
D. The disease is chronic and recurring
E. Patients often respond to anticholinesterase drugs

32. Guillian-Barré patients are challenging to ventilate continuously because:

I. They cannot communicate verbally
II. They are often long term ventilator patients
III. Their lungs are non-compliant

IV. They have problems with low blood sugar

A. I and II
B. II and III
C. I, II and III
D. I, II and IV
E. II, III and IV

33. The most common cause of pulmonary edema is:

A. Near drowning
B. Rapid IV fluid infusion
C. ARDS
D. COPD
E. Cardiac disease

34. The definitive diagnosis of respiratory failure is established through:

A. A chest x-ray
B. Clinical assessment
C. Patient mental status
D. Patient history
E. Arterial blood gases

35. When initiating mechanical ventilation, the tidal volume should be set at:

A. 5 ml/kg body weight
B. 10 ml/kg body weight
C. 15 ml/kg body weight
D. 18 ml/kg body weight
E. 22 ml/kg body weight

36. Which of the following criteria indicate that a ventilator patient can tolerate weaning?

I. PIP of 10-20 cm H_2O
II. Spontaneous minute ventilation greater than 10 lpm
III. Vital capacity greater than 10-15 ml/kg body weight

IV. V_D/V_T ratio less than 0.55

A. I and III
B. II and III
C. II and IV
D. III and IV
E. I, II and IV

37. Which of the following factors is the main cause of malnutrition in the chronically ill patient in the ICU setting?

A. Increased losses of nutrients
B. Increased metabolism
C. Inadequate absorption
D. Inadequate intake
E. Inadequate utilization

38. Which of the following procedures is the most reliable and accurate means of assessing the caloric requirements of the ventilator patient?

A. Daily weighing
B. Arm circumference determinations
C. Mobility of extremities
D. Basal metabolic rate determination
E. Indirect calorimetry

39. The most common means by which air enters the intrapleural space from within the lung is due to:

A. Atelectasis
B. Pulmonary fibrosis
C. Ruptured blebs
D. Pores of Kohn
E. ARDS

40. The definitive diagnosis of a pneumothorax is made by:

A. The presence of dyspnea
B. Arterial blood gases
C. A chest x-ray
D. Chest auscultation
E. Observing the respiratory pattern

41. Which of the following breath sounds would be associated with a hemothorax?

A. Dull
B. Resonant
C. Hyper-resonant
D. Vesicular
E. Bronchial

42. The lack of fluctuation in the water column in a closed-chest drainage system could be due to which of the following?

I. A kink in the tubing
II. Fluid in the pleural space
III. Air in the pleural space
IV. Clots in the chest tube

A. I and III
B. I and IV
C. II and IV
D. I, II and III
E. I, II, III and IV

43. Which of the following is/are clinical or pathophysiologic conditions in which an intra-aortic balloon pump may be indicated?

I. Aortic regurgitation
II. Peripheral vascular disease
III. Post-cardiopulmonary by-pass
IV. Cardiogenic shock

A. II only
B. III only
C. III and IV
D. I, II and III
E. II, III and IV

44. What is the primary physiological effect of intra-aortic balloon pumping?

A. Decrease in peripheral vascular resistance
B. Increase in arterial oxygenation
C. Reduction in arrhythmias
D. Increased contractility of the myocardium
E. Increased mean arterial pressure

45. What is the main criteria for determining whether a patient can be weaned from an intra-aortic balloon pump?

A. Absence of cardiac arrhythmias with the pump off
B. Maintenance of an adequate mean arterial pressure with the pump off
C. Maintenance of a normal cardiac rate with the pump off
D. Absence of a peripheral cyanosis with the pump off
E. Maintenance of an adequate acid-base status with the pump off

46. Extracorporeal membrane oxygenation is indicated when:

A. The patient has a P_aO_2 less than 55 mmHg on 80% oxygen
B. PEEP levels greater than 15 cmH_2O are required to maintain a P_aO_2 greater than 55 mmHg
C. The chest x-ray shows complete opacification of lung fields
D. The cardiac output is inadequate to maintain oxygenation of the tissues
E. There is severe chest wall dysfunction

47. The venovenous route for administering extracorporeal membrane oxygenation would have cannulas in which of the following vessels?

A. Femoral vein and femoral artery
B. Internal jugular vein and femoral artery
C. Both cannulas in the femoral artery
D. Innominate artery and femoral vein
E. Internal jugular vein and femoral vein

48. What is a major disadvantage of the venovenous route of administering extracorporeal membrane oxygenation??

A. All of the blood from the membrane oxygenator flows through the lungs
B. The blood from the membrane oxygenator is not evenly distributed throughout the body
C. A reduction of the right ventricular output
D. Ballooned cannulas cannot be used
E. Blood from the membrane oxygenator is mixed with venous blood

49. Which of the following are hazards associated with extracorporeal membrane oxygenation?

I. Pulmonary oxygen toxicity
II. Thrombotic phenomena
III. Uncontrolled bleeding
IV. Sepsis

A. I and II
B. II and III
C. I, II and IV
D. II, III and IV

E. I, II, III and IV

50. The primary criteria for weaning from extracorporeal membrane oxygenation is:

A. A reduction in the opacification of the chest x-ray
B. Maintenance of a stable P_aO_2 when the oxygenator blood flow is reduced
C. Stabilization of the clotting time as the oxygenator blood flow is reduced
D. Maintenance of the cardiac output as the oxygenator blood flow is reduced
E. Absence of peripheral cyanosis as the oxygenator blood flow is reduced

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