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# The hemodynamics of pulmonary and medical intensive care

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***Objectives:*** **To describe a) the physiology of oxygen delivery, b) the importance of hemodynamic monitoring in the medical intensive care unit, especially as it relates to pulmonary disease, c) methods for proper use of pulmonary artery catheters and interpretation of derived data and d) application of these principles to specific pulmonary diseases.**

***Oxygen Transport:*** **Fundamental to interpreting and responding to changes in hemodynamic data is having an understanding of oxygen transport and consumption, Frank-Starling's law, chemical mediators, mixed venous oxygen tension, continuous venous oximetry, and acid-base balance.**

***Hemodynamic Monitoring:*** **It is necessary to decide which patients will benefit from placement of a pulmonary artery catheter, select an insertion site and appropriate catheter, safely insert a catheter for measurement of cardiac, pulmonary artery and pulmonary artery occlusion pressure (wedge pressure), and interpret the resulting waveforms and pressure values. There are early and late complications of catheter placement that must be observed for.**

***Pulmonary Considerations:*** **Patients with pulmonary disease may have marked changes in intrathoracic pressure, pulmonary vascular resistance, cardiac output, and oxygen delivery to the tissues. In respiratory failure right ventricular performance can not be predicted from the right ventricular ejection fraction, and is better correlated with the right ventricular end-diastolic volume index.**

***Mechanical Ventilation:*** **An important adjunct to the treatment of critically ill patients, mechanical ventilation serves to improve oxygenation and reduce the work of breathing. Changes in intrathoracic pressure associated with the underlying pulmonary disease, PEEP, and type of ventilation all influence hemodynamic measurements. The most common and important hemodynamic effect of mechanical ventilation is to decrease cardiac output by decreasing the pressure gradient for venous return.**

***Pharmacologic Intervention:*** **Medications useful in the treatment of critically ill patients have significant hemodynamic effects, including influencing myocardial contractility, vascular resistance, preload and afterload.**

***Specific Disorders:*** **Pulmonary edema, chronic obstructive pulmonary disease, adult respiratory distress syndrome, pulmonary embolism, barotrauma and contusion are among the conditions often encountered, and provide a special challenge in obtaining, interpreting and responding to hemodynamic measurements.**

## OXYGEN TRANSPORT

Adequate tissue oxygenation depends on hemoglobin concentration, the percentage of hemoglobin saturated with oxygen in arterial blood ($SaO_2$), cardiac output (CO), oxygen consumption ($VO_2$), the affinity of hemoglobin for oxygen (P50) and the distribution of perfusion.

Normal compensatory mechanisms are typically

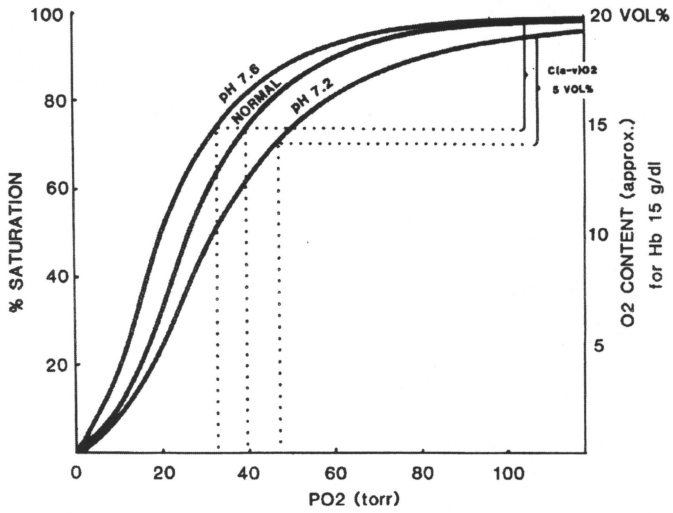


**Figure 1.** Oxygen dissociation curve [Snyder 1987].

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disturbed in critically ill patients, and shifts in the oxyhemoglobin dissociation curve (Fig. 1) such as from acidosis or alkalosis may profoundly impact oxygen content and delivery.

The curve may be shifted to the right with improved oxygen unloading (decrease in the affinity of hemoglobin for oxygen) at the tissue level by increased blood temperature, carbon dioxide, hydrogen ion concentration, 2,3-DPG, intercellular sodium, and hemoglobin concentration.

The curve may be shifted to the left by hypothermia, hypocarbia, alkalosis, anemia, and decreases in 2,3-DPG or sodium.

Although methods exist for approximating oxygen consumption in critically ill patients, they are technically difficult and may not give clue to individual organ metabolism. In summary, it is best simply to maximize oxygen transport to support the greater than normal metabolic rates.

Tissue oxygenation depends on both oxygen saturation and rate of flow:

$$TO_2 = CO \times CaO_2$$

$TO_2$ is oxygen transport, CO is cardiac output and $CaO_2$ is arterial oxygen content.

If cardiac output is severely depressed despite improvement in arterial oxygenation, oxygen transport may be worsened. It is necessary to consider all factors that contribute to oxygen supply and not rely solely on the level of arterial oxygen saturation improvement [Edwards 1993], [Snyder 1987].

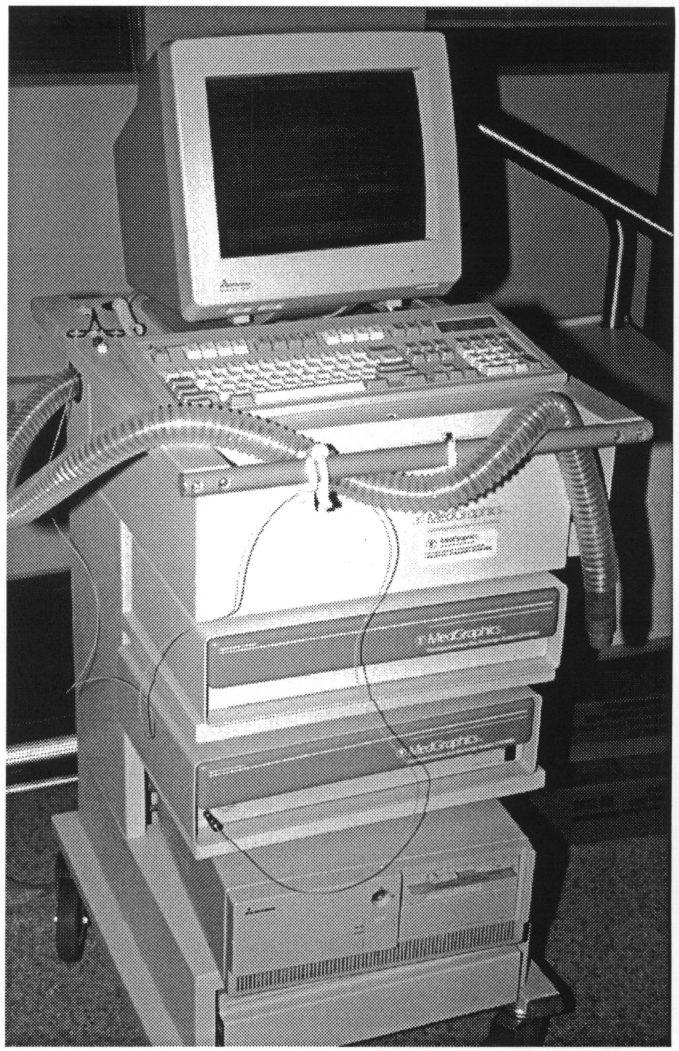

**Figure 2.** The MedGraphics Gas Exchange® system.

## Oxygen Consumption ($VO_2$)

The amount of oxygen utilized is normally determined by the body's energy requirements, and can be calculated from the Fick equation [Dantzker 1991]:

$$VO_2 = VI \times FIO_2 - VE \times FEO_2 = CO \times (CaO_2 - CvO_2)$$

VI and VE are the inspired and expired minute ventilations, $FIO_2$ and $FEO_2$ are the inspired and expired fractional concentrations of oxygen, CO is the cardiac output, and $CaO_2$ and $CvO_2$ are the arterial and mixed venous oxygen content.

In a steady state condition the amount of oxygen taken up by the tissues is equal to the amount taken up in the lung, so that $V0_2$ can be calculated from either the gas side of the system (Fig. 2), measuring the difference between the amount of oxygen in the inspired and mixed expired gas, or the blood side as the product of the cardiac output and the arterial-venous oxygen difference.

## Frank-Starling's Law

Cardiac output is dependent on heart rate, intrinsic myocardial contractility, preload and afterload .

The Frank-Starling curve (Fig. 3) shows the relationship between left ventricular (LV) preload and stroke volume (SV). For a given heart rate, as venous return increases, preload increases, and the force of ventricular contraction increases, allowing the heart to empty to a nearly constant end-diastolic volume or increased stroke volume. This is Starling's law, and only cardiac myofibrils - not skeletal or smooth muscle - have the ability to increase their force of contraction as fiber length increases above resting length.

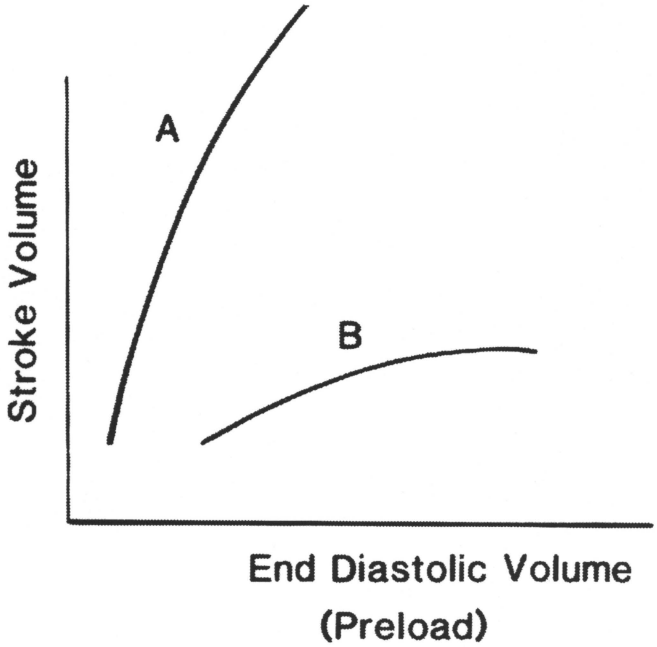


**Figure 3.** Frank-Starling Curve [Snyder 1987].

The ventricle described by curve A has a better performance than that described by curve B.

Pulmonary artery occlusive pressure (PAOP) is used as an estimate of end-diastolic pressure which in turn approximates LV preload [Goldberg 1987].

### Prostaglandins, Prostacyclin, Thromboxane A2 and Leukotrienes

Prostaglandins and related substances come from the breakdown of tissue, and are involved in producing shock, fever, and pain.

Prostacyclin modulates vasoconstriction in the microvasculature and participates in the hyperemic response to hypoxia. It augments renal flow, sodium and water excretion, and modulates hemostasis. The balance between prostacyclin and TXA2 is critical to maintaining hemostasis, and imbalance may cause vasospasm, infarction, and angina.

The leukotrienes are potent mediators of coronary vasoconstriction and produce negative inotropic effects [Bruns 1987], [Gerrard 1987], [Zeid 1987].

### Pulse Oximetry, Mixed Venous Oxygen Tension, Continuous Venous Oximetry

Pulse oximetry is commonly used for measuring arterial oxygen saturation (SaO2) (Fig. 4).

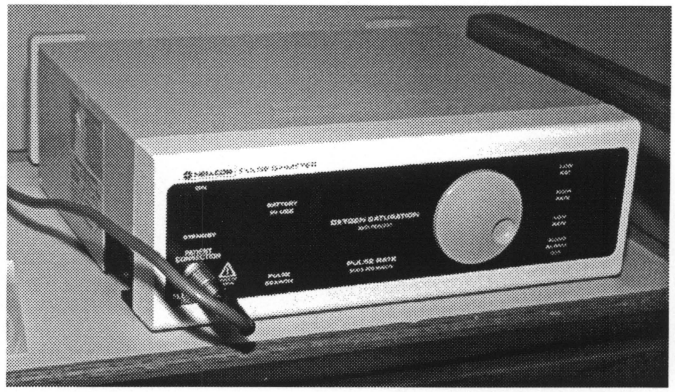

**Figure 4.** The Nellcor® Pulse Oximeter.

Mixed venous oxygen tension ($PVO_2$) may be the most reliable single physiologic indicator for monitoring the overall balance between oxygen supply and demand.

Mixed venous blood is a flow-weighted mixture of all blood that has traversed the systemic vascular beds and may best be sampled from the proximal pulmonary artery.

Marked venous hypoxemia ($PVO_2 < 27$ mmHg) and lactic acidosis is associated with high mortality.

Mixed venous oxygen tension does not indicate if a specific organ is under perfused or the distribution of perfusion. Another drawback is that in critically ill patients the central venous sample may not represent a true mixed venous sample.

Fiberoptic catheters for continuous analysis of blood oxygen saturation ($SvO_2$) has increased in popularity since first introduced in 1972 (Fig. 5). The $SvO_2$ reflects the overall balance between oxygen supply and demand. Calibration with a mixed venous sample and catheter position are important.

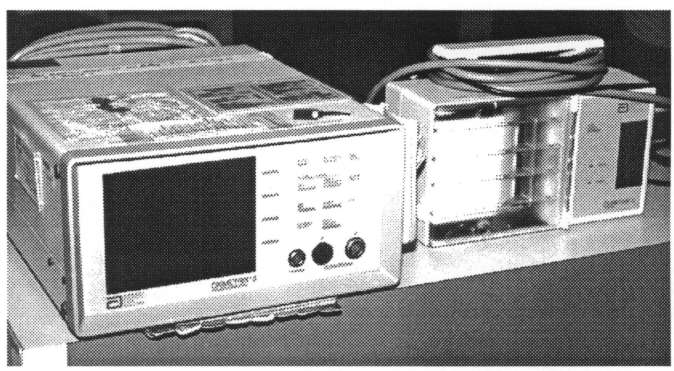

**Figure 5.** The Abbott Oximetrix® for measuring continous mixed venous oxygen saturation.

**Table 1.** Interpreting Arterial Blood Gases.

| Condition | Findings | Cause |
|---|---|---|
| **Respiratory Acidosis** | **(PaC02 > 45) + (pH < 7.35)** | **Inadequate ventilation** |
| **Metabolic Acidosis** | **(PaC02 < 35) + (pH < 7.35)** | **H+ buildup** |
| **Respiratory Alkalosis** | **(PaC02 < 35) + (pH > 7.45)** | **Increased ventilation** |
| **Metabolic Alkalosis** | **(PaC02 > 45) + (pH > 7.45)** | **Volume depletion or low K+** |

The normal range is 0.68-0.77. High values indicate an increase in delivery relative to consumption, and is associated with cirrhosis, sepsis, peripheral left-to-right shunting, cyanide toxicity, arterial hyperoxia or technical problems (calibration error, wedging of the catheter).

Low values may be associated with anemia, arterial oxygen desaturation, increased oxygen consumption or decreases in cardiac output. A rapidly falling $Sv0_2$ may proceed a major cardiovascular complication.

Interpreting arterial blood gases for acid-base balance is also important, and Table 1 allows for distinguishing respiratory from metabolic disorders [American Heart Association 1987].

[Martin 1992], [Nelson 1987], [Nelson 1992], [Snyder 1987], [Vincent 1992].

### Echocardiography

Comprehensive cardiovascular ultrasound imaging can be used to determine hemodynamics, including gradient, pressure, flow and valve area.

Since 1987 transesophageal echocardiography has been considered a routine extension of the precordial or transthoracic echocardiographic examination.

Superior results are obtained in prosthetic heart valves, mitral regurgitation, critical illness following infarct or trauma, endocarditis, cardiac tumors, thrombosis and masses, congenital heart disease and aortic disease [Seward 1991].

## HEMODYNAMIC MONITORING

The pulmonary artery catheter allows measurement of right atrial, right ventricular, pulmonary artery, occlusion (wedge) pressure, and cardiac output. Taken together with blood pressure, arterial and mixed venous oxygen content, the clinician can properly evaluate cardiopulmonary function, and through repeated measurements be able to monitor the patient's progress and response to therapeutic interventions.

Guidelines for catheter use and clinical competence in hemodynamic monitoring have been published [American Society of Anesthesiologists 1993], [Expert Panel 1991], [Friesinger 1990], [Naylor 1993], [Society of Critical Care Medicine 1992], [Young 1990].

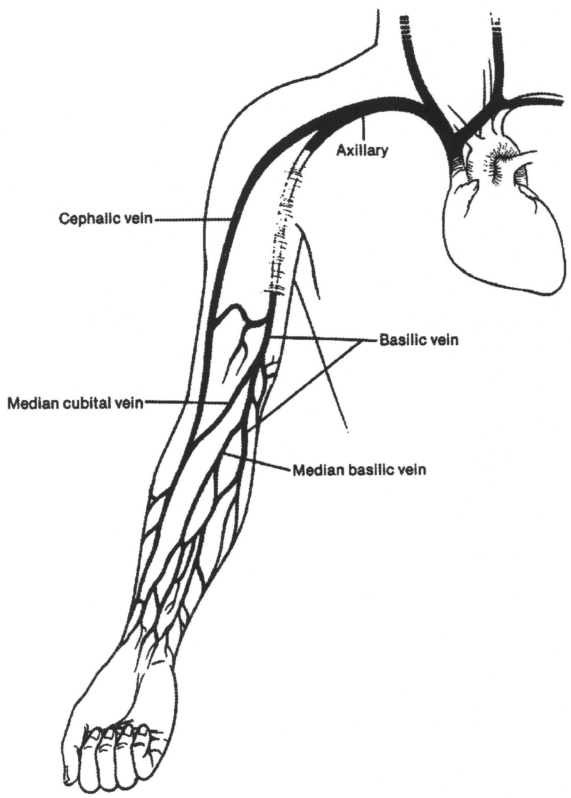


**Figure 5.** Anatomy of the arm veins [Daily 1989].

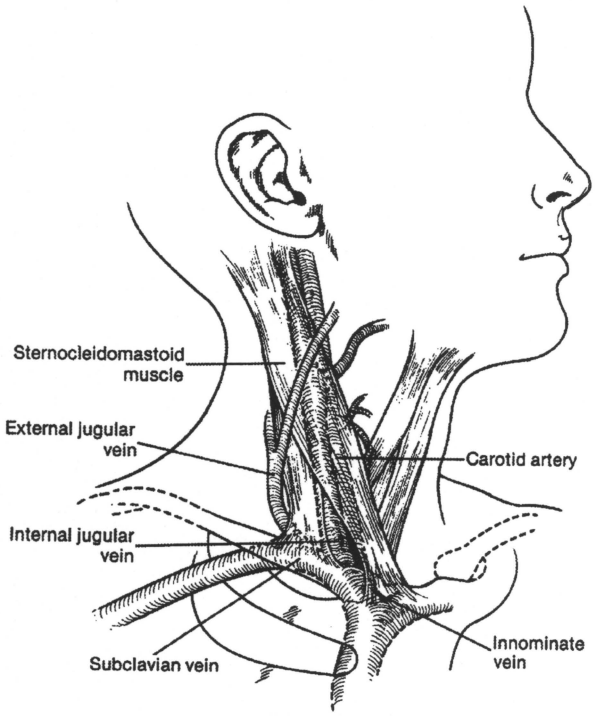


**Figure 6.** Anatomy of the Subclavian and Jugular Veins [Daily 1989].

## Insertion Technique

Common sites for catheter insertion include the medial basilic vein (Fig. 5), internal jugular vein and subclavian vein (Fig. 6). Femoral vein catheterization has also been shown to be safe and effective.

A ready-made tray, such as the Arrow® Percutaneous Sheath Introducer Kit speeds the process of venous access and catheter placement.

The site (Fig. 7) is first cleansed and draped, and then using an 18 Ga. x 2.5 catheter assembly, the vein is located. A plastic sheath remains in place, while an inner metal needle is removed. A 0.035 inch spring wire is inserted through the sheath, and then the sheath is also removed.

A vessel dilator is pre-placed through a detachable hemostasis valve and larger flexible sheath, and the combination assembly is slid down the wire into the vein. The wire and dilator are removed, and an intravenous solution is connected to the side-port of the valve.

A pulmonary artery catheter, such as a Swan - Ganz® type is removed from its container and the balloon inspected after inflation for symmetry. Enough air should be used to recess the tip of the catheter. The air is passively deflated and the catheter is wiped clean with saline.

The catheter is placed through the contamination

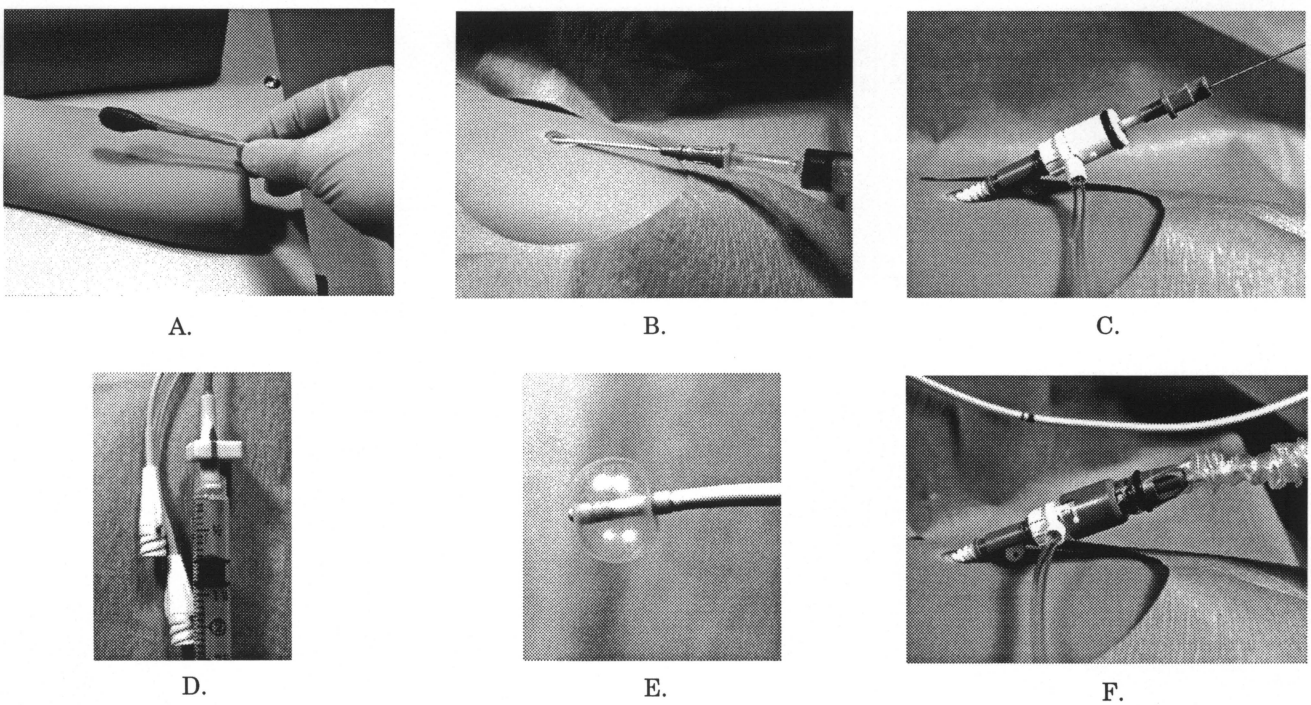


**Figure 7.** Catheter insertion: a) skin preparation, b) vein location and placement of a plastic sheath, c) advancement of a larger sheath, hemostasis valve and vein dilator down the spring wire, d) use of a safety syringe, e) testing for balloon function and f) placement of the pulmonary artery catheter and contamination shield.

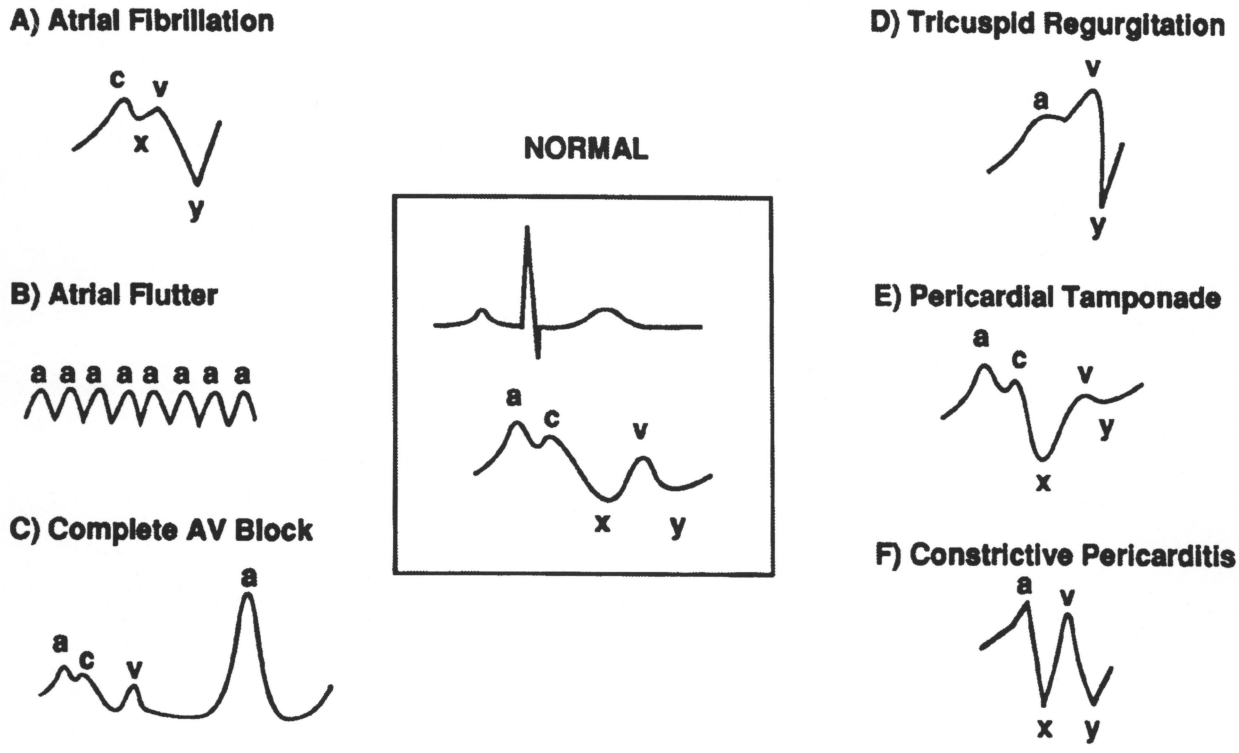


**Figure 8.** Representative right atrial pressure waveforms [Civetta 1992].

shield first, then slid into the hemostasis valve. The manometer is connected so that waveforms can be interpreted as the catheter is advanced.

The balloon should not be inflated until in the superior vena cava or right atrium. Advance until a pulmonary artery occlusive pressure reading is obtained, then deflate the balloon and pull it back into the pulmonary artery.

The hemostasis plug should be sutured to the skin and dressed. The contamination shield is unfolded providing a margin of safety for intermittently re-advancing the catheter and checking the pulmonary occlusive pressure reading.

### Hemodynamic Pressure Measurement and Tracings

The *a* wave is due to atrial systole and follows the P wave of the electrocardiogram (Fig. 8). The *c* wave occurs with tricuspid valve closure. The *x* descent is due to a combination of atrial relaxation and the downward displacement of the atrioventricular junction during the early part of ventricular systole. The *v* wave corresponds to the flow of blood into the atria against a closed tricuspid valve during the late part of ventricular systole. The *y* descent results from the rapid flow of blood from the atria into the ventricles following opening of the mitral and tricuspid valves. Additional examples of waveforms are shown in Figs. 9-14.

The *a* wave is absent in patients with atrial fibrillation (A).

Flutter *a* waves are observed in patients with atrial flutter (B).

Large *a* waves ("Cannon *a* waves") occur when the atria contract while the atrioventricular valves are closed during ventricular systole. These may be regular in junctional rhythm or irregular when atrioventricular dissociation accompanies premature ventricular beats, ventricular tachycardia or complete heart block (C).

With tricuspid insufficiency, the right atrial *v* wave becomes prominent, the *x* descent is obliterated, and the *y* descent is steep (D).

Pericardial tamponade causes elevation and equalization of the right atrial, pulmonary artery diastolic, and wedge pressure tracings. The *x* descent on the right atrial pressure tracing is preserved; however the *y* descent is damped or absent due to restricted early ventricular filling (E).

Constrictive pericarditis also causes elevation and equalization of diastolic pressures. The *a* and *v* waves are followed by prominent *x* and *y* descents, resulting in a typical "M" waveform. Unlike in tamponade, the

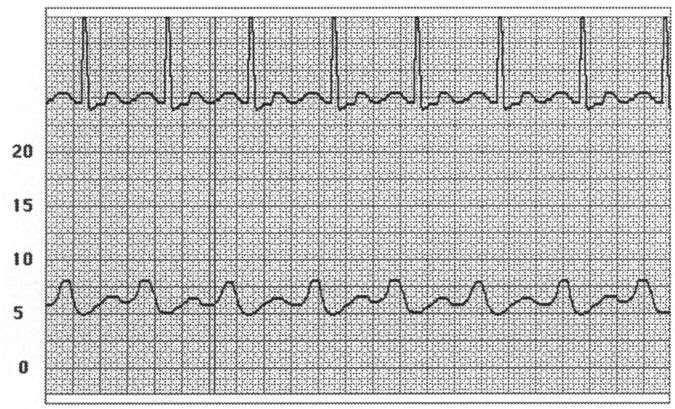


**Figure 9.** Normal right atrial pressure. *All tracings are from the Dynacath Critical Care Patient Simulator™.*

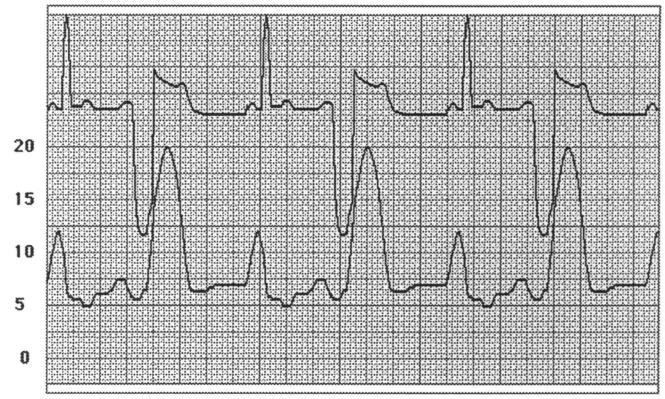


**Figure 12.** Ventricular bigeminy. Note the Cannon *a* wave with each premature ventricular contraction.

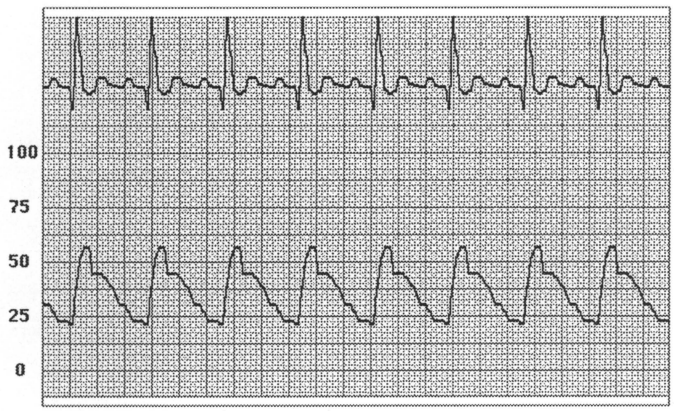


**Figure 10.** Normal pulmonary artery pressure.

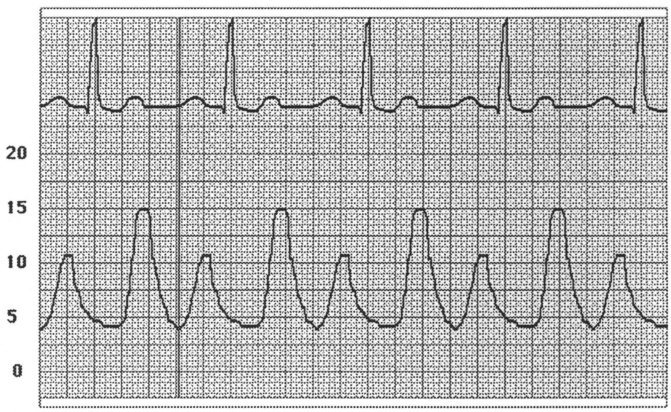


**Figure 13.** Tricuspid insufficiency. Typically the large *v* wave occurs early in diastole.

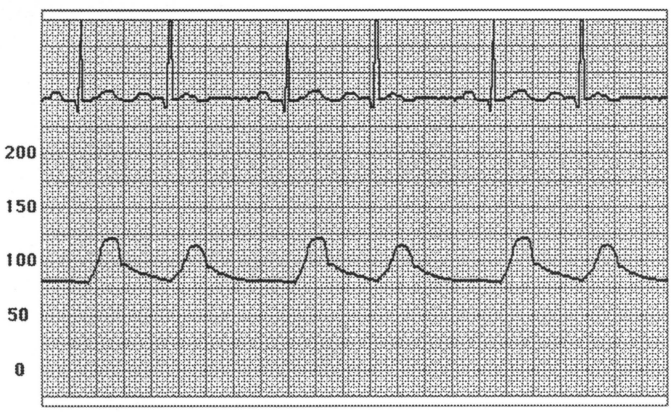


**Figure 11.** Premature atrial contractions. Note the arterial pressure variation with shorter filling time.

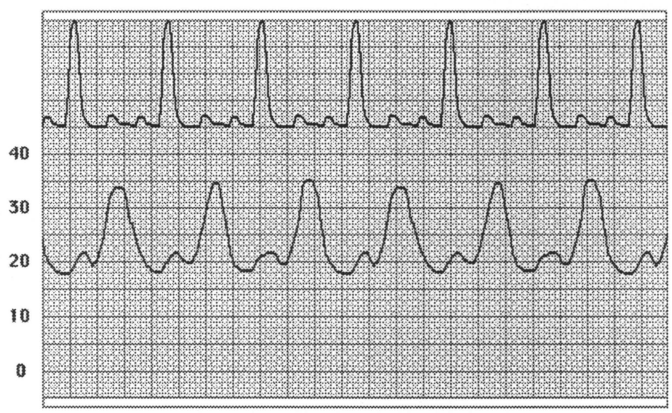


**Figure 14.** Mitral insufficiency. Typically the large *v* wave occurs late in diastole.

*y* descent is prominent because there is no restriction of early ventricular filling (F).

In acute mitral insufficiency, a large *v* wave may be present in the pulmonary artery wedge tracing. This has been attributed to the regurgitant blood flow across the incompetent mitral valve into a relatively noncompliant left atrium. The *v* wave may mimic the pulmonary artery waveform and be misinterpreted as the catheter being in the pulmonary artery position. Compare with the ECG. The peak of the *v* wave occurs after the T wave (the peak of the pulmonary artery systole occurs within the T wave). Mitral obstruction, congestive heart failure and VSD may also have large v waves.

With right ventricular failure, the right ventricular end-diastolic pressure may be so high that during catheter insertion the right ventricular waveform may resemble the pulmonary artery tracing.

In hypovolemic shock, extremely low right ventricular and pulmonary artery pressures may be observed.

[Ahrens 1991], [Astiz 1993], [Bach 1992], [Berlauk 1991], [Biga 1991], [Bridges 1993], [Cobean 1992], [Connors 1992], [Cope 1992], [Ermakov 1992], [Ferraris 1992], [Findling 1994], [Hamilton-Farrell 1990], [Her 1993], [Iberti 1992], [Masood 1989], [Nolan 1992], [Ornato 1993], [Pagliarello 1993], [Roth 1992], [Shively 1991], [Shoemaker 1990], [Sola 1993], [Spodick 1989], [Steingrub 1991], [Tuchschmidt 1994], [Tuman 1989], [Vine 1991], [Weed 1991], [West 1992], [Yelderman 1992], [Zion 1990].

## Complications of Hemodynamic Monitoring

Complications of central venous and pulmonary artery cannulation include the following:

1. Immediate Complications:
   a. Multiple puncture
   b. Pneumo/hemo/hydro/chylothorax-mediastinum
   c. Arterial puncture - hematoma or bleeding
   d. Air embolism
   e. Cardiac arrhythmias
   f. Catheter malposition
   g. Catheter knotting
   i. Subcutaneous and mediastinal emphysema
   j. Tracheal puncture-laceration

2. Late Complications:
   a. Pulmonary artery rupture
   b. Pulmonary infarction
   c. Catheter-related sepsis
   d. Balloon rupture
   e. Endocardial or valvular damage
   f. Venous thrombosis
   g. Infection
   h. Nerve injury
   i. Cerebrovascular compromise
   j. Cardiac perforation and tamponade
   j. Arteriovenous fistula
   k. Thrombocytopenia

[Allyn 1989], [Baraka 1991], [Bernardin 1994], [Duong 1993], [Feng 1990], [Gotchall 1989], [Guillaume 1990], [Hagley 1992], [Keusch 1989], [Kirton 1992], [McLellan 1989], [Mermel 1991], [Mermel 1993], [Mermel 1994], [Moorthy 1991], [Raad 1993], [Smart 1990], [Soding 1994], [Unger 1990], [Venus 1992], [Westenskow 1993].

## Is Hemodynamic Monitoring of Value?

Inadequate training in pulmonary artery catheter placement and interpretation may account for the diversity of opinion as to the benefits of invasive monitoring [Iberti 1990]. In addition, physician assessment of the effect of right heart catheterization on treatment decisions and patient outcomes has been challenged [Ontario Intensive Care Study Group 1992]. There may also be interobserver variability in the interpretation of tracings [Komedina 1991]. For the present, it is believed that properly obtained data may direct the physician toward the most appropriate therapy, and improve patient outcome.

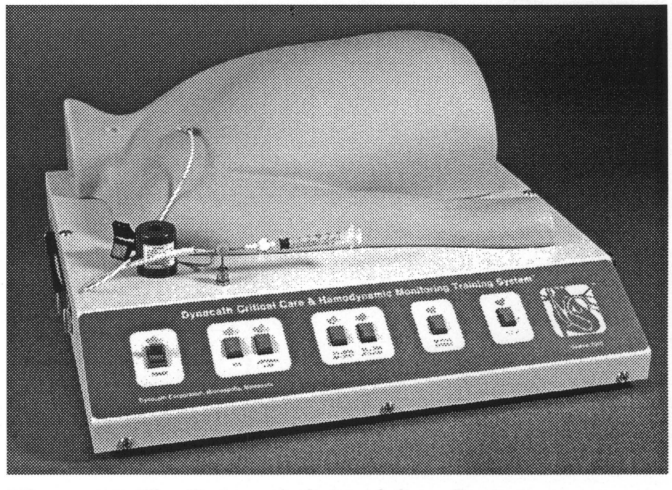


**Figure 15.** The Dynacath Critical Care Patient Simulator™.

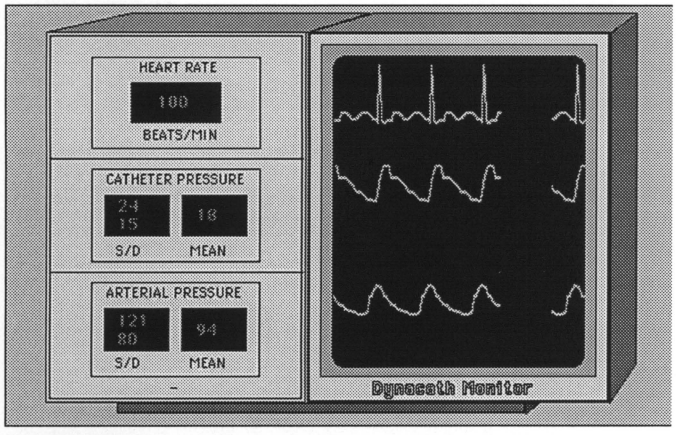


**Figure 16.** Apple Macintosh® Computer display of a cardiac monitor, with simulated patient data from the Critical Care Patient Simulator™.

### Improved Training

The Critical Care Patient Simulator (Figs. 15-16) may be used to practice pulmonary artery catheter placement, interpret waveforms and integrate simulated hemodynamic data with case scenarios [Saliterman 1990]. This system also allows a trainee to order studies, select management plans and observe the hemodynamic consequences of therapeutic intervention.

## PULMONARY CONSIDERATIONS

### Ventilation/Perfusion

The physiology of ventilation and perfusion is helpful in understanding changes that occur with pulmonary disease. The determinants of local ventilation are local compliance, local airway resistance, and local change in transepithelial pressure. Compliance in normal lungs depends on relative alveolar volume (gravity dependent). Normally ventilation of the lung base is greater. In low lung volumes the proportion of gas delivered to upper lung units is greater. Airway resistance is dependent on edema, interstitial elasticity, bronchospasm, and obstruction (collapse or mucous).

Flow is diminished in the nondependent lung by gravity and compression of the alveolar vessels if PAOP is increased. Because of interstitial pressure, flow is decreased in the most dependent lung [Snyder 1987].

### Pulmonary Vascular Resistance

Pulmonary vascular resistance (PVR) may be calculated by the following:

$$(PAP - PAOP)/CO \times 80$$
(normal 100-250 dynes•sec•cm-5)

The following may be associated with increased PVR [Fromm 1987]:

1. Primary Pulmonary Hypertension.
2. Vasoconstriction caused by hypoxemia and aci dosis.
3. Pulmonary obstructive processes - severe COPD or PE.

### Right Atrial Pressure

Patients with pulmonary disease frequently have marked swings in intrathoracic pressure, and hemodynamic pressure measurements parallel these changes. There may be excessive fall in pressures during spontaneous inspiration.

The RA pressure may be normal in mild pulmonary disease or moderately elevated in severe disease.

Elevated RA pressures resulting from pulmonary disease denote increased right-heart dysfunction caused by increased pulmonary vascular resistance (PVR) or afterload. The a wave of the RA waveform becomes more dominant.

Wide swings in the RA pressure may occur with the respiratory cycle, and it is important to obtain a mean RA pressure at end-expiration over three to four cycles [Daily 1989].

### Right Ventricular Function

The right ventricle is essential to cardiopulmonary function. Failure of the right ventricle is the inability to maintain adequate distribution of blood flow to ventilated lung segments [Nelson 1993].

In acute respiratory failure, the right ventricular ejection fraction does not reflect right ventricular performance [Her 1993]. There may be a severe reduction in right ventricular ejection fraction while the right ventricle continues to generate sufficient pressure for pulmonary perfusion.

This is because the ejection fraction is dependent on the afterload, and afterload may change dramatically in patients with acute respiratory failure. An

increase in pulmonary vascular resistance increases the afterload of the right ventricle.

The right ventricular end-systolic pressure volume relationship may be a clinically useful tool to assess right ventricular contractile function.

Right ventricular end-diastolic volume index is calculated as the stroke volume index (cardiac index/heart rate) divided by ejection fraction. This correlates better with cardiac output than do either the central venous pressure or pulmonary artery occlusion pressure.

A low value predicts that volume loading will likely increase the cardiac index in critically ill patients. A high value (>140 ml/$m^2$) implies that volume loading will be unlikely to improve cardiac index [Diebel 1992].

These values are less likely to be affected by positive end-expiration pressure (PEEP) than pressure-based variables [Eddy 1993], [Cheatham 1993].

Right ventricular end-diastolic volume may be measured using the Baxter Explorer System® pulmonary artery catheter.

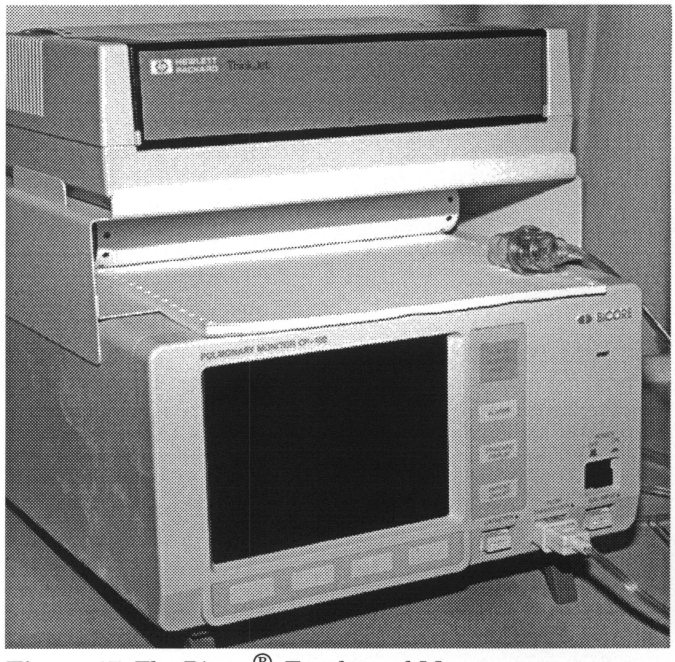


**Figure 17.** The Bicore® Esophageal Manometer system.

### Pulmonary Artery Pressure

The PAP change depends on the degree of change of PVR and/or CO.

Pressure = Flow x Resistance

Increased PVR may be offset initially by decreased flow ("low cardiac output pattern"). More typically in the critical care patient the PVR will be sufficiently high to cause the PAP to be high.

Normally PA end-diastolic pressure (PAEDP) equals the PAOP or LAP, and reflects the LVP at end-diastole. This allows following the PAEDP rather than obtaining the PAOP pressure.

In pulmonary disease with increased PVR, the PAP may become elevated while the PAOP or LAP is low. There are two reasons for this:

1. When the balloon is inflated, flow stops in the pulmonary capillary, and measured pressure is a reflection of the LAP.
2. Marked reduction of lung compliance in pulmonary disease often prevents the transmission of increased pleural fluid pressure to the pulmonary microvasculature.

An increase in PVR is the major limiting factor in CO in patients with pulmonary disease. The arteriovenous oxygen difference is abnormally wide.

The SvO2 is frequently lower than normal as a result of increased oxygen extraction as a compensation to decreased oxygen delivery.

In general, because of the wide swings in intrathoracic pressure, accurate hemodynamic pressure readings can be measured by obtaining an end-expiratory pressure reading and averaging over three-four respiratory cycles. This may require use of a paper write-out rather than the scope display.

### Esophageal Pressure (Pes) Monitoring

Esophageal pressure monitoring allows measurement of fluctuations in global intrathoracic pressure (Fig. 17). Pes allows estimation of the force generated during all patient-initiated breaths (spontaneous or mechanically ventilated), and allows partitioning of lung and chest wall components. Knowledge of the Pes helps interpret PAOP pressure during PEEP. Changes in Pes can be used to determine the work of breathing.

A thin esophageal catheter (about 2mm in size) with a long balloon is inflated, passed into the stomach, and then withdrawn until negative pressure deflections are observed during spontaneous inspiratory efforts. The balloon is withdrawn another 10 cm, and its final position tested by occluding the airway and measuring deflections in PAOP and Pes. These should closely approximate one another [Marini 1989].

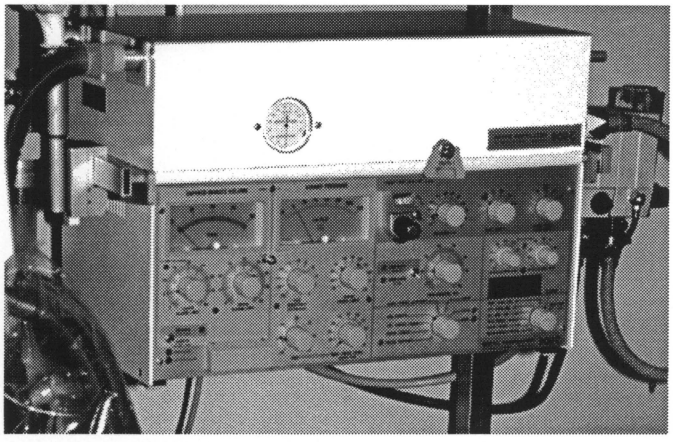

**Figure 18.** The Siemens Servo Ventilator.

## MECHANICAL VENTILATION

Mechanical ventilation is used to improve oxygenation, treat alveolar hypoventilation and reduce the work of breathing (Fig. 18). Normally only 5% of total oxygen consumption goes to the work of breathing. In respiratory distress this can increases to as much as 50%. Muscle fatigue, observed by tachypnea, recruiting of accessory muscles, paradoxical muscle motion, discoordination and finally respiratory failure, may be relieved by mechanical ventilation.

The heart, existing in the thorax, is a pressure chamber within a pressure chamber. Therefore changes within the thorax (intrathoracic pressure - ITP) will affect the pressure gradient for blood returning to the chest (venous return), and leaving the chest (left ventricular output).

Whereas ITP falls with spontaneous inspiration, it rises with positive pressure ventilation (Fig. 19).

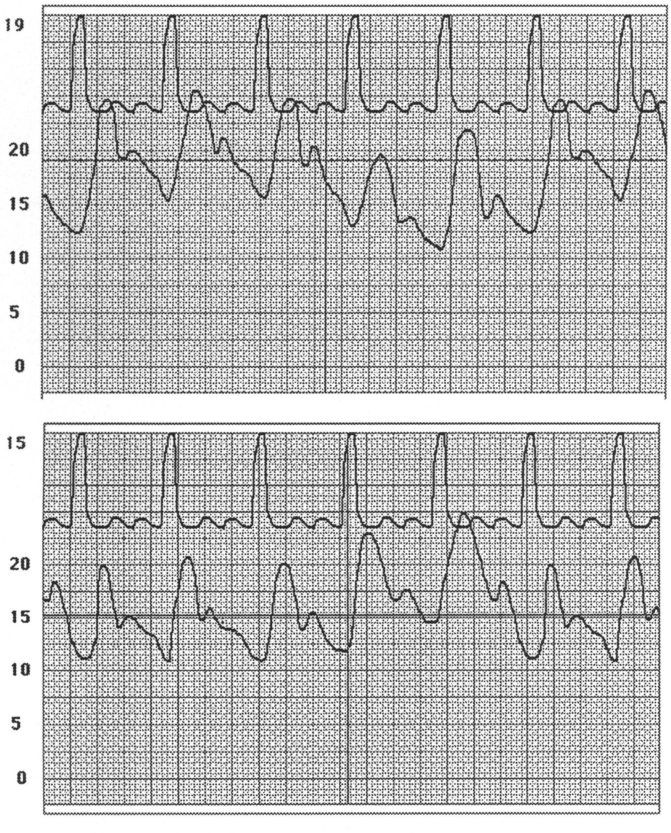


**Figure 19.** End-expiration occurs about the middle of each tracing above. The upper strip shows normal pulmonary artery pressure with spontaneous inspiration. The negative ITP produces a negative swing in the tracing. The lower strip shows a positive swing from increased ITP associated with mechanical ventilation.

### Mode of Ventilation

Common modes of ventilation include assist control (ACV), synchronized intermittent mandatory (SIMV) and pressure support (PSV). Hemodynamic and oxygen transport pararmeters in patients on these various forms of ventilation have been studied [Sternberg 1994].

SIMV and PSV are commonly used for weaning. The PSV augments the patient's spontaneous breaths with a preset positive pressure delivered by the ventilator. The patient controls the respiratory rate, inspiratory flow and time. It reduces the ventilatory work done by the patient and improves the breathing pattern and patient's comfort.

All three modes increase intrathoracic pressure (ITP), lower venous return and lessen cardiac output. During the spontaneous breaths in SIMV the ITP is lower, and cardiac output (CO) increases. By allowing lower peak and mean airway pressures throughout the respiratory cycle SIMV marginally improves the cardiac index (CI) over ACV.

There is a slightly higher CI, oxygen transport and oxygen consumption on SIMV and PSV. It is felt that SIMV and PSV when used for 30 minutes can provide adequate ventilation with lower airway pressure and possibly less adverse effects on hemodynamic and tissue oxygenation parameters compared with ACV.

### Positive Pressure Ventilation

Continuous positive airway pressure (CPAP) provides positive expiratory and inspiratory pressure in spontaneous breathing. When applied to pressure breathing it is referred to as positive end-expiratory

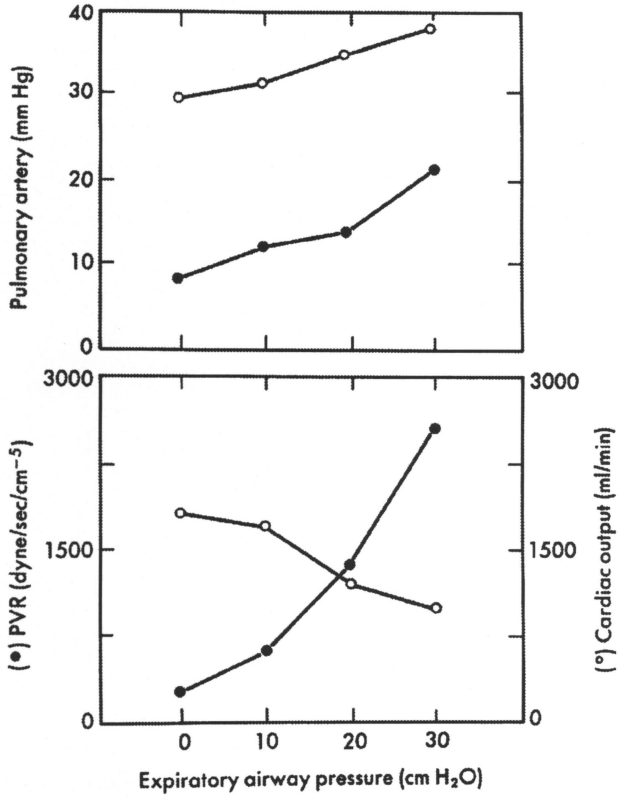


**Figure 20.** Changes in pulmonary artery pressure (PA), pulmonary vascular resistance (PVR) and cardiac output (CO) with increases in positive end-expiratory pressure (PEEP) (Daily 1989).

pressure (PEEP). Acute respiratory failure is often associated with a decrease in functional reserve capacity (FRC), and CPAP or PEEP may help decrease PVR (expanding collapsed areas) - improving venous return and LV filling.

As PEEP is increased, PVR and PAP increase, while CO decreases (Fig. 20).

### Estimating Transmural Pressure

Since hemodynamic measurements obtained with mechanical ventilation are altered by increases in intrathoracic pressure, attempts may be made to estimate transmural pressure. Turning off the respirator for measurement can be dangerous. Distal esophageal balloons or inserting a catheter in the pleural space can allow determination of pleural pressure, but is not widely done.

Mean intrathoracic pressure is normally -3 mmHg. The true transmural pressures therefore would really be the measured pressure minus the estimated intrapleural pressure.

PAOP - Pleural Pressure = Transmural PAOP
(eg. 8 mmHg - (-3 mmHg) = 11 mmHg)

Normally this small and constant factor can be ignored in patients not mechanically ventilated. The extent to which the intrapleural pressure is increased depends on the mode of ventilation.

PEEP therapy increase intrapleural pressure only about 1/3 of the applied airway pressure because critical care patients usually have lungs that stiff and non-compliant. Effects or PEEP can be ascertained by measurement of the PAOP when PEEP is momentarily discontinued for suctioning.

### Responding to the Effects of Mechanical Ventilation

Hemodynamic monitoring is helpful in patients receiving mechanical ventilation to achieve a satisfactory balance between the beneficial effect of increased oxygenation and the deleterious effects of decreased cardiac output.

When low blood pressure and cardiac output is due to decreased venous return, consider administering fluids, elevating the legs, pressure suit, minimizing increases in ITP by decreasing inspiratory time, decreasing tidal volume (Vt) or using the minimum amount of PEEP.

Volume loading may be beneficial if the right ventricular end-diastolic volume index is low.

A low CO with normal or high transmural PAOP or LAP may be secondary to LV dysfunction - such as can occur from hypoxemia - and further fluids would be adverse. An inotropic agent, such as dopamine, dobutamine, and epinephrine may be necessary to improve contractility.

The left ventricular function curve compares LV performance (either CO or stroke volume) as a function of LV filling pressure (LVEDP or PAOP) (Fig. 21). As the LVEDP increases, CO increases (Frank-Starling law). Increases in PAOP over 20 mmHg usually produce little or no improvement in CO. The "A" arrow and curve reflects a shift to a higher ventricular function curve and stroke volume without a change in preload, as seen with positive inotropic therapy. The "B" arrow reflects a shift to a higher ventricular function curve and stroke volume at a lower preload - as occurs with vasodilator therapy. The "C" curve reflects a shift to a lower preload and stoke volume while remaining on the same ventricular function curve, as occurs with diuretic therapy.

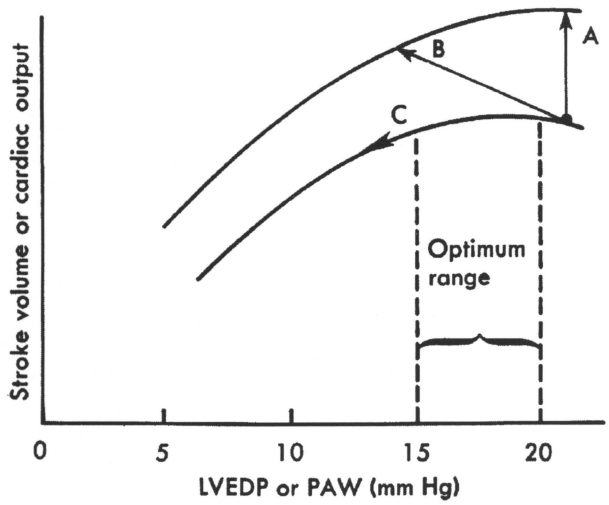


**Figure 21.** Left ventricular function curves, showing improvement with a) positive inotropic agents, b) vasodilators and c) diuretics (Daily 1989).

As CO falls reflex vasoconstriction (to maintain perfusing BP) increases SVR, afterload. and hence further reduces CO. Vasodilating agents or afterload-reducing agents such as nitroprusside or nitroglycerin. Indications are low CO, normal to high PAOP LAP with high SVR.

### Clinical Application

When cardiac function is normal, venous return is the primary determinant of cardiac output. Increases in RAP induced by positive pressure ventilation will decrease cardiac output by increasing back pressure to venous blood flow.

A decrease in venous return will especially be pronounced in hypovolemic states (hemorrhage, dehydration), or when vasomotor tone is decreased (sepsis, spinal shock, autonomic blockage).

A decrease in venous return induced by positive pressure breathing may be responsible for cardiovascular collapse seen in some patients immediately after endotracheal intubation and "bagging" for acute respiratory failure.

Hemodynamic measurements may not be accurate in these patients - the PAOP may not accurately reflect LV filling when hyperinflation or high levels of PEEP (>12 cm $H_2O$). In such settings other parameters of cardiac output should be monitored such as cardiac output, mixed venous oxygen saturation, arterial-venous oxygen differences and urine output.

Sudden rises in PAP (hyperinflation, high PEEP, PE, hypoxic pulmonary vasoconstriction) can induce cor pulmonale. Venous return goes down, and the dilated RV impinges on the LV, decreasing compliance and filling, and further decreasing cardiac output. Countermeasures include bronchodilators, minimum inspiratory time ratio, minimum PEEP, and in most cases, fluid administration to increase RV filling pressure.

[Ahrens 1991], [Daily 1989], [Ishizawa 1989], [Lookinland 1989], [Pery 1991], [Pinsky 1987], [Poelaert 1991]

## PHARMACOLOGIC INTERVENTION

### Norepinephrine

Norepinephrine is a potent alpha receptor agonist which causes arterial and venous vasoconstriction, and beta-1-agonist which increases myocardial contractility. It increases BP because of increased SVR, and may not improve or worsen CO. It is a useful temporizing measure in hemodynamically significant hypotension refractory to other sympathomimetic amines. Usual does is 2-12 mcg/min.

### Dopamine

Dopamine in low doses (1-2 mcg/k/min) produces vasodilation of renal, mesenteric and cerebral arteries. AT medium doses of 2-10 mcg/kg/min there is enhanced cardiac output and only a modest increase in SVR. At doses above 20 mcg/kg/min alpha-adrenergic effects predominate with constriction of vasculature and increases SVR much like norepinephrine, Dopamine is useful when there is hemodynamically significant hypotension in the absence of hypovolemia. Combined with vasodilators, such as nitroprusside, effects similar to dobutamine are achieved - that is improved myocardial contractility with reduced preload and improved cardiac output.

### Dobutamine

Dobutamine is a potent inotropic agent that stimulates beta-1 and alpha-adrenergic receptors in myocardium. Its minor stimulation of peripheral alpha receptors is antagonized by more potent beta-2 stimulation, and mild vasodilation may occur. It does not produce renal or mesenteric vasodilation via dopaminergic receptors, improves balance between oxygen supply and demand and does not induce

arrhythmias. It is useful in the treatment of pulmonary congestion and low cardiac output or in the hypotensive patients in whom vasodilators cannot be used because of intolerance to lower pressures. It is the treatment of choice along with volume loading in patients with hemodynamically significant right ventricular infarction. Usual doses is 2.5-20 mcg/kg/min.

### Isoproterenol

Isoproterenol is a beta-adrenergic inotropic and chronotropic medication which increase cardiac output in spite of peripheral vasodilation, venous pooling and reduction in mean blood pressure. It increase myocardial oxygen requirements and worsens ischemia. It is useful for hemodynamically significant atropine refractory bradycardia. It can cause serious arrhythmias. Usual dose is 2 mcg/min.

### Digitalis

Digitalis is used to increase myocardial contraction and control ventricular response to atrial fibrillation and flutter. It has little role in the management of acute congestive heart failure.

### Sodium Nitroprusside

Sodium nitroprusside is a potent peripheral vasodilator with effects on both arterial and venous smooth muscle. It is useful in the emergency treatment of hypertension and heart failure by reducing blood pressure and preload (via decreased peripheral vascular resistance from increased venous capacitance). May be useful when heart failure and pulmonary congestion is acutely or poorly controlled by diuretic therapy. It is the treatment of choice for hypertensive emergencies, and is easily titratable. Usual starting dose is 0.5 mcg/kg/min titrated to desired endpoint.

### Nitroglycerin

Nitroglycerin relaxes smooth muscle, relieves angina, reduces LV filling pressure and SVR, decreases oxygen requirements and has the net effect of increasing cardiac output. It is useful in the emergency treatment of congestive heart failure, especially in patients with ischemic heart disease. Tolerance may develop, and intermittent dosing with nitrate-free periods may be beneficial. Usual dose is 10-20 mcg/min.

### Propranolol

Propranolol is a nonselective agent which attenuates the effects of circulating catecholamines by blocking their ability to bind to beta receptors. It has both cardiac and pulmonary effects. Its primary emergency indication is for control of recurrent ventricular tachycardia, recurrent ventricular fibrillation and/or supraventricular arrhythmias refractory to other therapy. Adverse effects include precipitation of hypotension, CHF and bronchospasm.

### Furosemide

Furosemide inhibits the reabsorption of sodium and chloride in the ascending loop of Henle. In patients with pulmonary edema during myocardial infarction, intravenous furosemide exerts direct venodilating effects that reduce venous return, and thus central pressure. These effects may be observed even more than that of diuresis. Usual dose is 20-40 mg IV up to 2.0 mg/kg total.

[American Heart Association 1987]

## SPECIFIC PULMONARY DISORDERS

### Pulmonary Edema

Hydrostatic and oncotic pressure gradients, in conjunction with barrier characteristics of alveolar epithelium, favor retention of fluid within the interstitial space (Fig. 22).

Pulmonary edema is often caused by a combination of increased intravascular pressure and increased capillary permeability. The latter the predominant cause of edema in sepsis or ARDS.

In severe LV failure, intrathoracic blood volume increases, and pulmonary edema may form. This occurs primarily because of increased pressure, but also in part from capillary leak. This decreases pulmonary compliance and gas exchange. Vasodilators, inotropic agents and diuretics, as well as addition of positive pressure breathing (including CPAP) may be necessary. This will decrease intrathoracic blood volume by decreasing venous return, and may improve cardiac performance by increasing ITP [Mecca 1992].

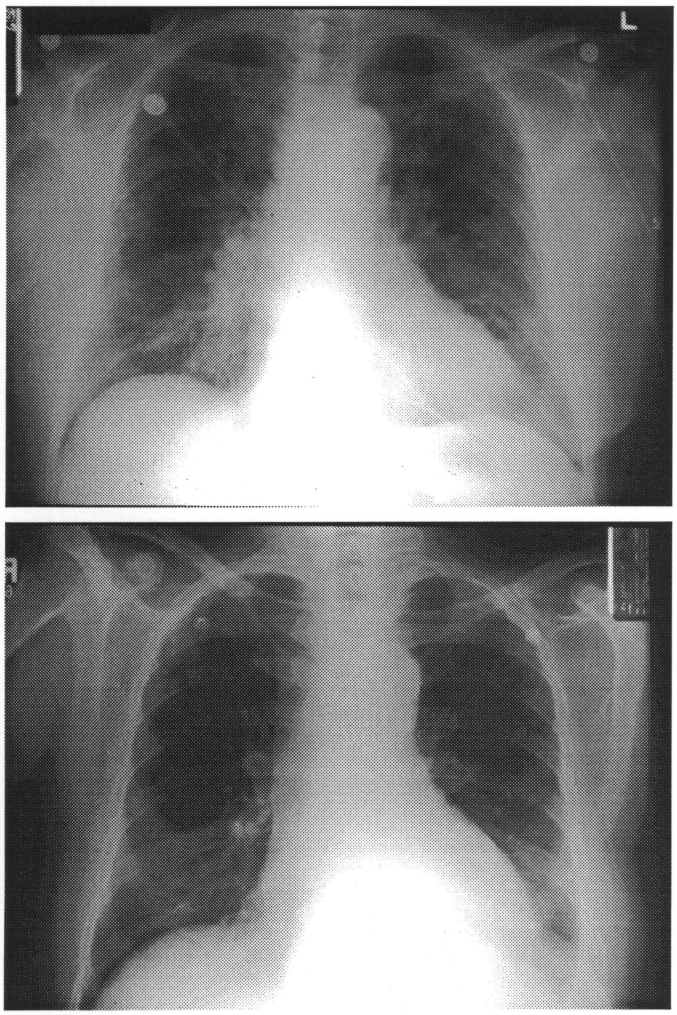

**Figure 22.** Pulmonary edema before treatment is shown in the upper film, and following diuresis in the lower film.

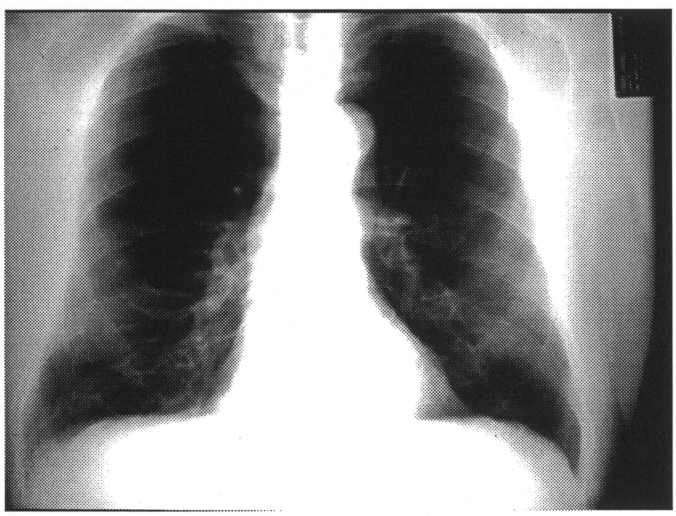

**Figure 23.** Chronic obstructive pulmonary disease (COPD). Note the hyperlucent lung fields, and flattened diaphragms.

### Chronic Obstructive Pulmonary Disease

Chronic obstructive pulmonary disease (COPD) includes chronic bronchitis, emphysema, asthma, bronchiectasis and cystic fibrosis.

Emphysema is characterized by abnormal enlargement of the airspaces distal to the terminal and respiratory bronchioles. There are destructive changes of the alveolar walls and capillary membranes. The chest x-ray often shows flattening of the diaphragms (Fig. 23).

Acute respiratory failure is associated with alveolar hypoventilation and elevation of the arterial partial pressure of carbon dioxide ($PaCO_2$). Patients with acute respiratory failure superimposed on chronic respiratory failure can be distinguished from patients with chronic respiratory failure alone by decreased arterial pH and slightly higher than normal sodium bicarbonate. In chronic respiratory failure the kidneys retain bicarbonate resulting in a low normal arterial pH, increased bicarbonate and decreased serum sodium chloride.

COPD is characterized by impaired gas exchange from ventilation/perfusion inequalities.

Exaggerated increases in intrathoracic pressure (> 20 mmHg) or alveolar pressure during expiration may be transmitted to the pulmonary microvasculature in COPD.

COPD patients with large chests may have inaccurate zero calibration of the transducer.

[Cicale 1992]

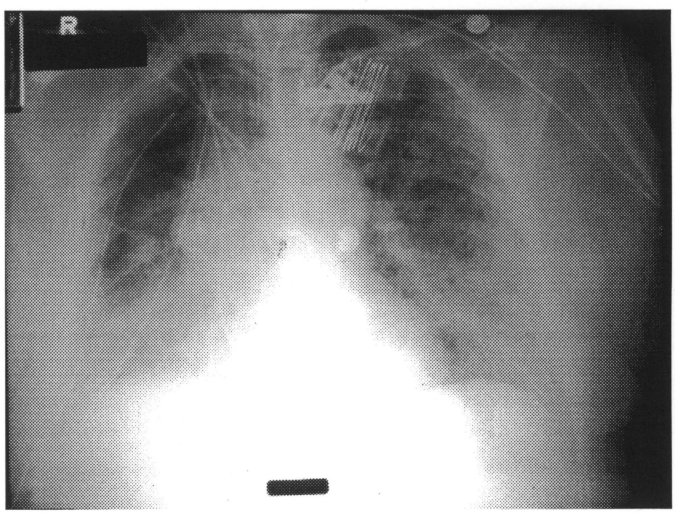

**Figure 24.** Adult respiratory distress syndrome (ARDS) following coronary artery bypass.

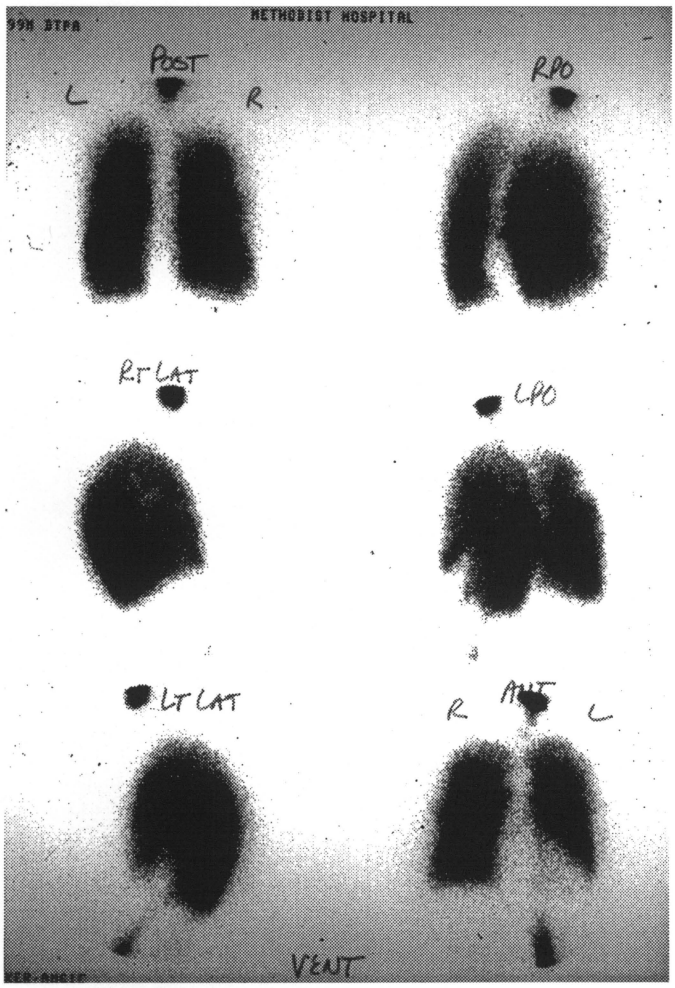


**Figure 25.** Normal ventilation scan.

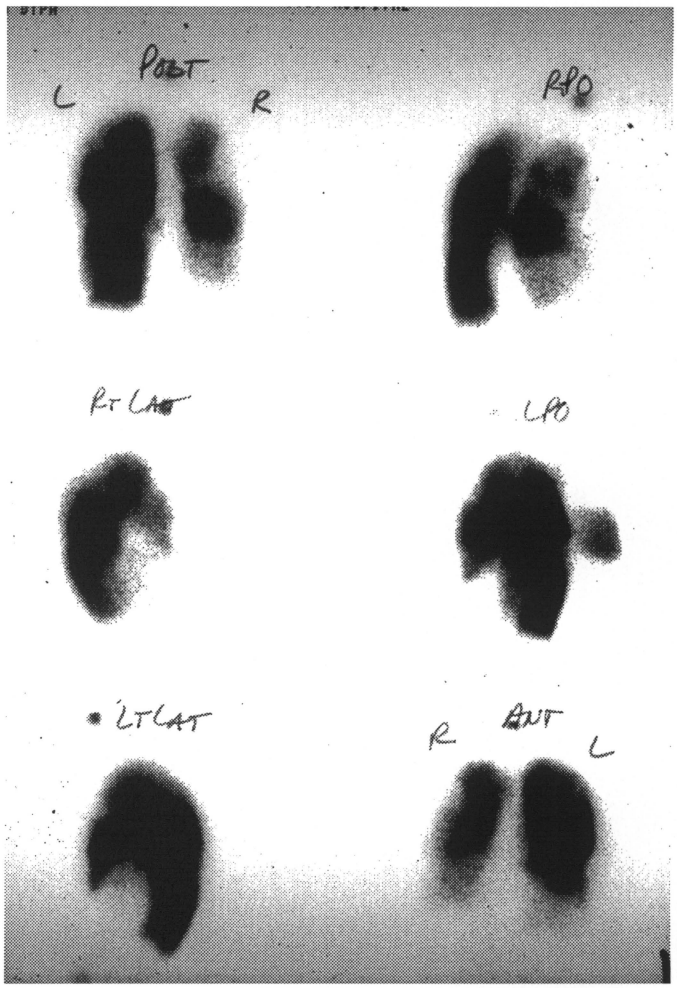


**Figure 26.** Abnormal perfusion scan, showing mismatched defects when compared to the prior ventilation scan.

### Adult Respiratory Distress Syndrome

Adult Respiratory Distress Syndrome [ARDS] is characterized by dyspnea, hypoxemia, diffuse bilateral pulmonary infiltrates and stiff lung. It is caused by diffuse lung injury that leads to increase in extravascular lung water (Fig. 24), usually occurs in patients with no previous lung disease and has an estimated mortality as high as 50% in some studies. Aspiration of gastric contents and sepsis are highly associated with ARDS. Other causes include shock, trauma, toxic gases, drug ingestion, metabolic (uremia, Diabetic ketoacidosis), pancreatitis, CAB, multiple transfusions, DIC, and eclampsia.

Interstitial and alveolar edema, fibrosis and surfactant abnormalities lead to reduced lung compliance (reduced functional residual capacity).

Treatment of the underlying etiology is important, because there is no way to stop the capillary leak and fibrosis.

PEEP is the most effective support therapy. It should be titrated in increments of 2 to 3 cm H20 every 15 mins. with reassessment of arterial oxygen tension, shunt fraction and oxygen transport.

Some volume expansion may be necessary to compensate for reduction in cardiac output associated with positive pressure ventilation or PEEP. If hemodynamic performance is acceptable, judicious diuresis may help decrease lung water. Excessive diuresis can be hazardous. Choice of fluid replacement is controversial - crystalloid (albumin) is acceptable [Taylor 1992].

### Pulmonary Embolism

Pulmonary embolism presenting symptoms include sudden onset of dyspnea, tachycardia, tachypnea, pleuritic chest pain, cough, fever, fatigue and apprehension.

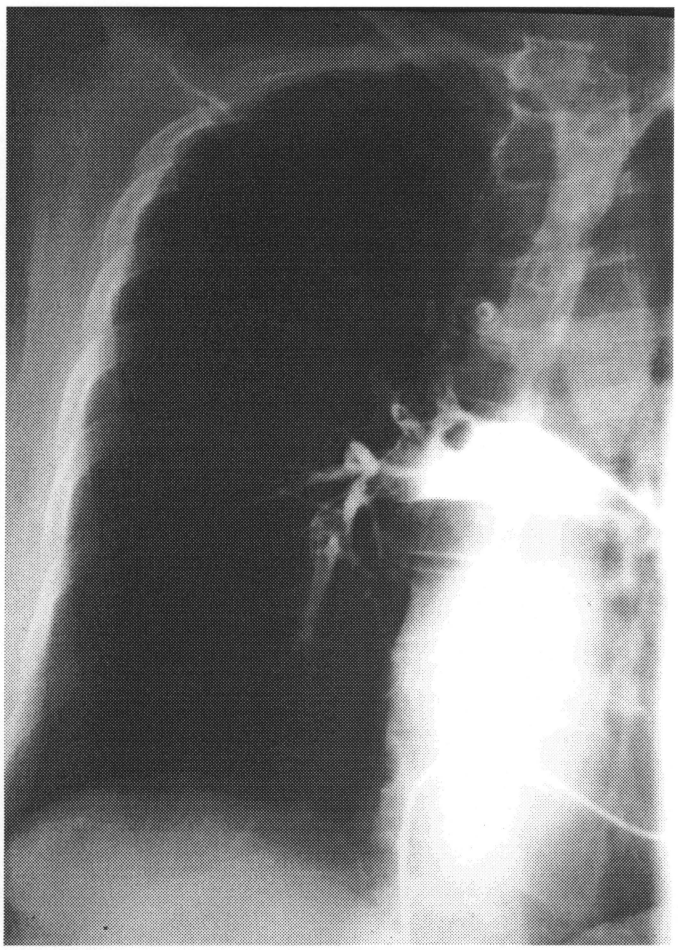

**Figure 27.** Angiogram showing segmental pulmonary artery occlusions, consistant with pulmonary embolism.

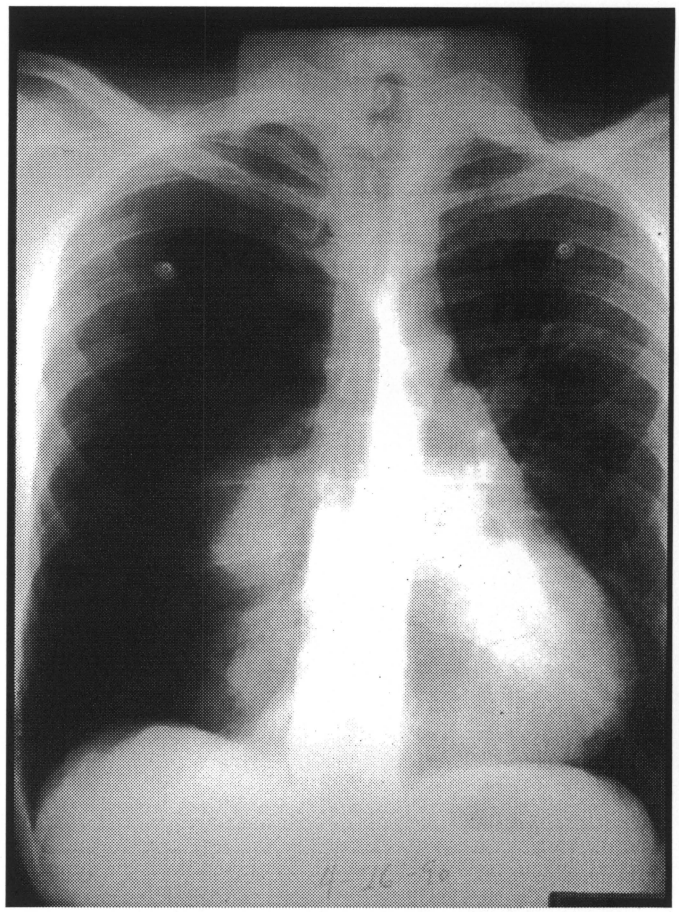

**Figure 28.** Spontaneous tension pneumothorax.

Acute cor pulmonale findings include right ventricular tap, augmented split pulmonic closure sound, right ventricular S3 gallop, distended neck veins, pulsus paradoxus (exaggerated fall in BP with inspiration), and Kussmaul's sign (paradoxic distention of neck veins with inspiration).

Arterial blood gases show hypoxemia with respiratory alkalosis.

Ventilation and perfusion scans (Figs. 25-26) are the best means of non-invasive diagnosis. Pulmonary angiography may be necessary to diagnose in patients with severe COPD (Fig. 27).

Treatment includes Heparin, oxygen, and in patients with massive embolism, isoproterenol may increase cardiac output and reduce pulmonary hypertension. If there is systemic hypotension, fluids and norepinephrine may be helpful. Morphine sulfate is useful in reducing pain and apprehension [Mandeep 1992].

## Barotrauma

Barotrauma is a complication of traumatic thoracic injury and therapeutic interventions. This includes pneumothorax, pulmonary interstitial emphysema, subcutaneous emphysema, pneumomediastinum. pneumopericardium, pneumoperitioneum and venous or arterial air embolization.

Pneumothorax is accompanied by hypoxemia resulting from increased intrapulmonary shunting, and reduction of cardiac output can occur if tension pneumothorax occurs. Decreased ventilation leads to hypercapnea and respiratory acidemia. Treat with chest tube is findings significant.

Tension pneumothorax findings include tachypnea/dyspnea, cyanosis, decreased blood pressure/cardiac output, hyperresonance to percussion ipsilateral side, and asymmetric chest movement with ventilation (Figs. 28-29).

Subclavian puncture and mechanical ventilation can cause pneumothorax. Incidence of this problem can be reduced by minimizing the number of mechanically delivered breaths, peak inspiratory pres-

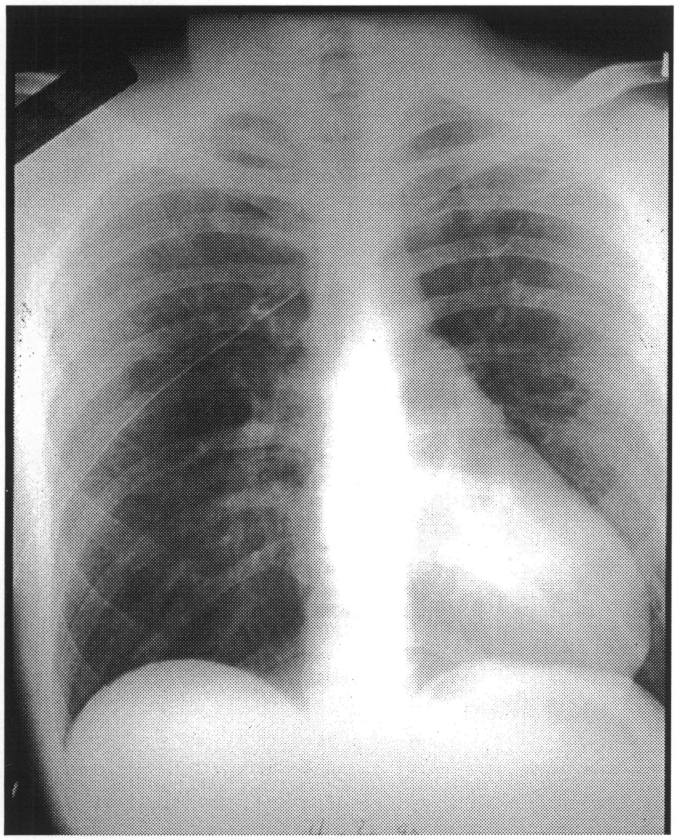

**Figure 29.** Pneumothorax treated with chest tube placement.

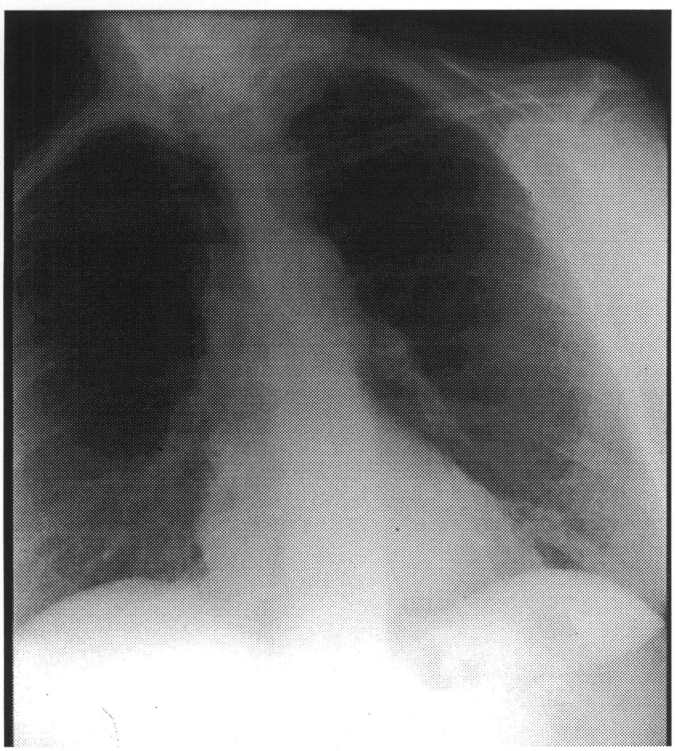

**Figure 30.** Contusion with development of a fluffy infiltrate in the right hemithorax.

sures, avoiding high PEEP/CPAP and maintaining intravascular volume [Brown 1992].

### Contusion

Contusion may be determined by a pulmonary infiltrate appearing 24 to 48 hours after rib fracture or flail chest, or may appear more subtle, such as progressive hypoxia without rib fracture or flail chest (Fig. 30). Hematomas often develop, or in severe cases, they may evolve into ARDS. Oxygen and CPAP may be helpful in mild cases, and in severe cases, fluids and crystalloids may be helpful. Management should be similar to ARDS above [Mecca 1992].

## SUMMARY

- It is necessary to consider all factors that contribute to oxygen saturation and not rely solely on the level of arterial oxygen saturation improvement.

- Mixed venous oxygen tension and continuous venous oximetry are important for judging the overall balance between oxygen supply and demand.

- Properly obtained hemodynamic data may direct the physician toward the most appropriate therapy, and improve patient outcome.

- Because of wide-swings in intrathoracic pressure, accurate hemodynamic pressure readings can be measured by obtaining an end-expiratory pressure reading and averaging over three or four respiratory cycles. This may require use of a strip chart recorder.

- In respiratory failure right ventricular performance can not be predicted from the right ventricular ejection fraction, and is better correlated with the right ventricular end-diastolic volume index.

- Mechanical ventilation is used to improve oxygenation, treat alveolar hypoventilation and reduce the work of breathing.

- The most common and important hemodynamic effect of mechanical ventilation is to decrease cardiac output by decreasing the pressure gradient for venous return.

- Estimating transmural pressure can be helpful in interpreting hemodynamic data in patients receiving mechanical ventilation.

• Hemodynamic monitoring in patients receiving mechanical ventilation is helpful in achieving a satisfactory balance between the beneficial effect of increased oxygenation and the deleterious effects of decreased cardiac output.

• Certain pulmonary disorders, like ARDS, especially if accompanied by severe left ventricular failure, require frequent assessment of hemodynamic and other data for proper guidance of therapy.

• Immediate and late complications of central venous and pulmonary artery cannulation should be monitored for.

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