Hemodynamic Monitoring

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Hemodynamic monitoring may be defined as the collection and interpretation of various parameters that inform determination of: (1) the etiology of a state of hypoperfusion and/or (2) the response of the cardiopulmonary unit to interventions such as fluid therapy, vasoactive drugs, or adjustments in positive pressure ventilation. For many patients, adequate monitoring is achieved by routine vital signs along with collection of data such as input/output, physical examination, and urine electrolytes. In other patients, invasive measurements are made, including use of arterial catheters, central venous catheters (CVC), and right heart catheters (RHC). These catheters provide for continuous transduction of pressure in either the arterial or venous circuit and sampling of blood for determination of oxygen saturation. Simultaneous determination of arterial and mixed venous blood gases also permits determination of oxygen content, oxygen delivery, oxygen consumption, arteriovenous oxygen content difference, and calculation of cardiac output by Fick determination.

The use of invasive methods of assessing hemodynamics—arterial and right heart catheters—grew during the evolution of critical care medicine despite a lack of prospective trials demonstrating efficacy and improved patient outcome. Indeed, one retrospective study suggested that use of the RHC is associated with an independent negative effect on survival. This study has been criticized largely on the basis of design—it was retrospective, and thus even reasonably sophisticated methods of case matching may have failed to control for the inevitable differences in patient status and hence prognosis that might contribute to decisions to perform invasive monitoring. However, prospective trials have been undertaken and one recent large multicenter study evaluating the use of RHC for high-risk surgical patients failed to demonstrate either a benefit or detriment to its use. It is important to note that in order for trials of invasive monitoring to demonstrate benefit, investigators must identify a patient population at risk for or exhibiting a hemodynamic state amenable to interventions that will improve outcome—monitoring alone is unlikely to confer benefit. Moreover, for many conditions—sepsis, the acute respiratory distress syndrome (ARDS)—the proper fluid and vasoactive drug interventions remain to be defined. For these conditions, trials have been designed and implemented that test not only the monitoring modality but the proper intervention as well (*eg*, randomizing patients with ARDS to either a RHC or CVC and then further to either a "fluid liberal" or "fluid conservative" management strategy).

Differential Diagnosis of Hypoperfused States and Bedside Assessment

A useful and readily applicable bedside algorithm at the time of resuscitation of patients with circulatory inadequacy is—is this low- or high-output hypotension? If the former, is the heart full or not? And when fluid resuscitation has occurred, is the response definitive or has low-output shock now taken on the characteristics of high-flow shock (*eg*, septic shock with initial hypovolemia, now fluid-resuscitated)? Often this simple algorithm succeeds in fully resuscitating the patient. If not, further information gathering from invasive monitoring and/or echocardiography is appropriate (Table 1).

Alternatives to Pulmonary Artery Catheterization

Given uncertainties concerning the benefits of invasive monitoring with the RHC, recent literature has emphasized alternative approaches. In the most significant recent trial evaluating resuscitation of patients with early severe sepsis and septic shock, RHC was not used but rather patients were randomized to routine care vs early goal-directed therapy (EGDT) guided by arterial blood pressure, right atrial pressure, right atrial oxygen saturation (as a surrogate of mixed venous blood saturation) and urine volume. Outcomes were improved with EGDT despite no use of RHC, suggesting the use of the cardiac output to determine the adequacy

of the circulation in patients with sepsis may be less useful than the concentration of effluent blood returning from the systemic circulation.

There has also been considerable study of the use of the arterial pressure waveform alone as an indicator for the adequacy of intravascular volume and response to fluid challenge. Numerous studies have shown that responders and nonresponders to fluid challenge are not well defined by the baseline right atrial pressure or pulmonary artery occlusion pressure (Figure 1). This relates to many factors to be discussed below.

However, patients with spontaneous respirations will typically exhibit drops in right atrial pressure during inspiration related to swings in intrathoracic pressure that has been shown to correlate with relative hypovolemia and "preload reserve," making this observation useful in determining the need for further fluid resuscitation. In addition, patients undergoing mechanical ventilation often have respiratory excursion of arterial blood pressure as demonstrated below (Figure 2).

The result of these cyclical changes in tidal volume is to cause a cyclical change in stroke

Table 1. *Rapid Formulation of an Early Working Diagnosis of the Etiology of Shock*

Defining Features of Shock			
Blood pressure	⇓		
Heart rate	⇑		
Respiratory rate	⇑		
Mentation	⇓		
Urine output	⇓		
Arterial pH	⇓		
		High Output Hypotension	*Low Cardiac Output*
		Septic Shock	*Cardiogenic and Hypovolemic*
Is Cardiac Output Reduced?		No	Yes
Pulse pressure		⇑	⇓
Diastolic pressure		⇑	⇓
Extremities digits		Warm	Cool
Nailbed return		Rapid	Slow
Heart sounds		Crisp	Muffled
Temperature		⇑ or ⇓	⇔
White cell count		⇑ or ⇓	⇔
Site of infection		++	-
		Reduced Pump Function	*Reduced Venous Return*
		Cardiogenic Shock	*Hypovolemic Shock*
Is the Heart Too Full?		Yes	No
Symptoms clinical context		Angina ECG	Hemorrhage dehydration
Jugular venous pressure		⇑	⇓
S_3, S_4, gallop rhythm		+++	-
Respiratory crepitations		+++	-
Chest radiograph		Large heart ⇑ upper lobe flow Pulmonary edema	Normal
What Does Not Fit?			
Overlapping etiologies (septic cardiogenic, septic hypovolemic, cardiogenic hypovolemic)			
Short list of other etiologies			
High output hypotension		*High right atrial pressure hypotension*	*Nonresponsive hypovolemia*
Liver failure		Pulmonary hypertension	Adrenal insufficiency
Severe Pancreatitis		(most often pulmonary embolus)	Anaphylaxis
Trauma with significant		Right ventricular infarction	Spinal shock
SIRS		Cardiac tamponade	
Thyroid storm			
Arteriovenous fistula			
Paget's disease			
Get more information		Echocardiography, right heart catheterization	

volume that is detectable on the arterial pressure waveform and signals the existence of hypovolemia (Figure 3).

Empiric investigation has shown that when a greater than 13% increase in the pulse pressure change between maximal (Ppmax) and minimal (Ppmin) pulse pressure exists, patients are highly likely to respond to fluid challenge:

$$\Delta PP\ (\%) = 100 \times ((Ppmax - Ppmin)/(Ppmax + Ppmin/2))$$

The receiver operator curve for the pulse pressure variation using this threshold and other measures or cardiac preload in patients with sepsis and hypoperfusion are shown in Figure 4.

While not validated on large groups of patients, this approach is attractive and could eventually prove to be more useful than measurements of right atrial or pulmonary capillary wedge pressure.

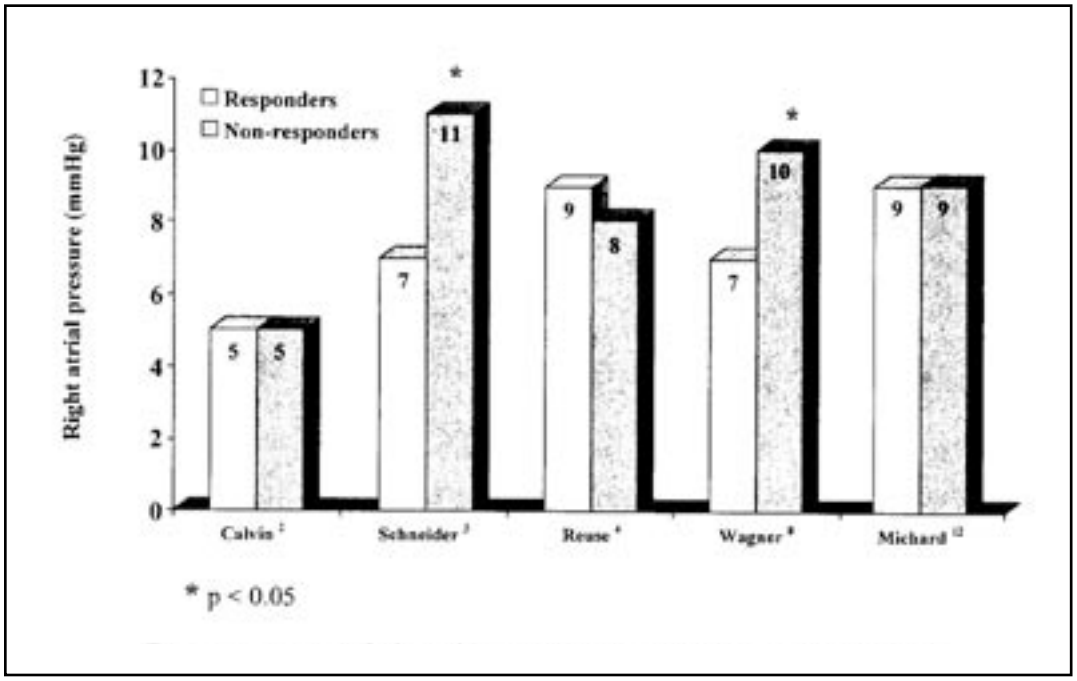

Figure 1. Mean MAP before volume expansion in responders and nonresponders.

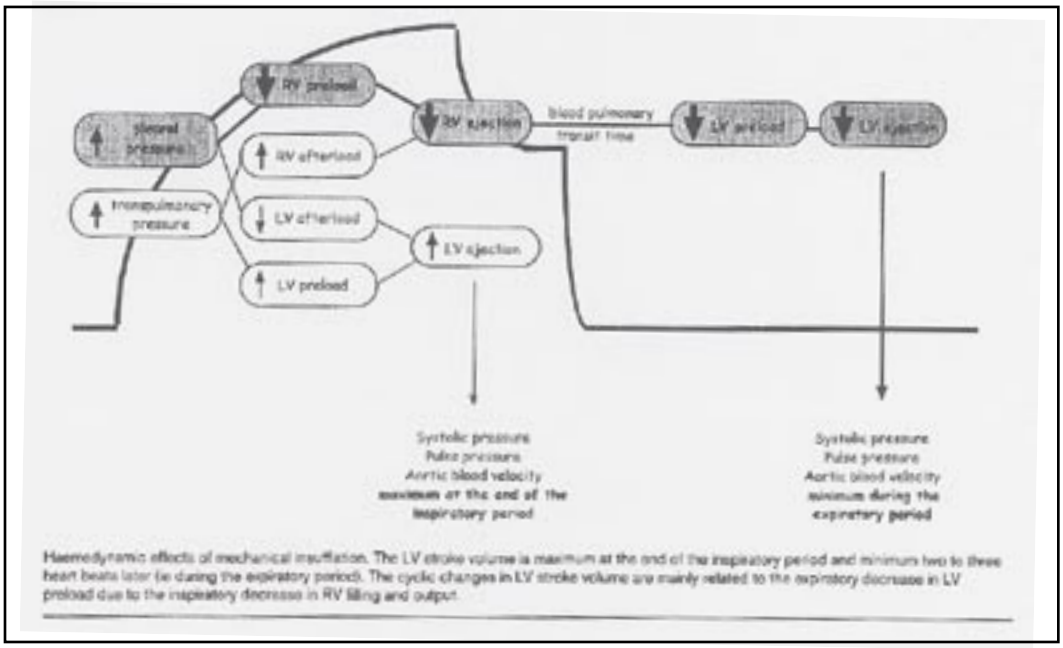

Figure 2.

Pulmonary Artery Catheterization

Indications and Complications

Rather than offer a list of many conditions that may require PA catheterization, the reader is guided to the statement above recommending formulation of questions concerning the etiology of hypoperfusion or the response to therapy, and answer these questions if possible with clinical data, including volume or drug challenges. When this approach is inadequate, PA catheterization is to be considered. Complications of the procedure are given in Table 2.

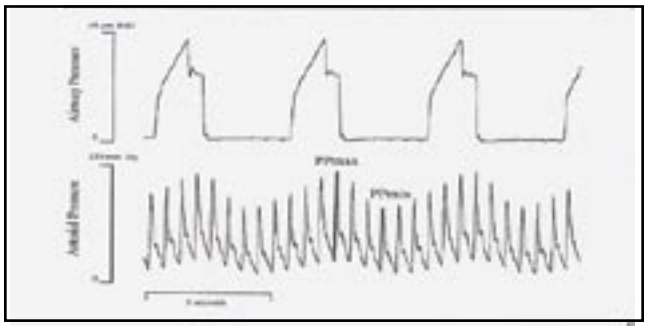

Figure 3.

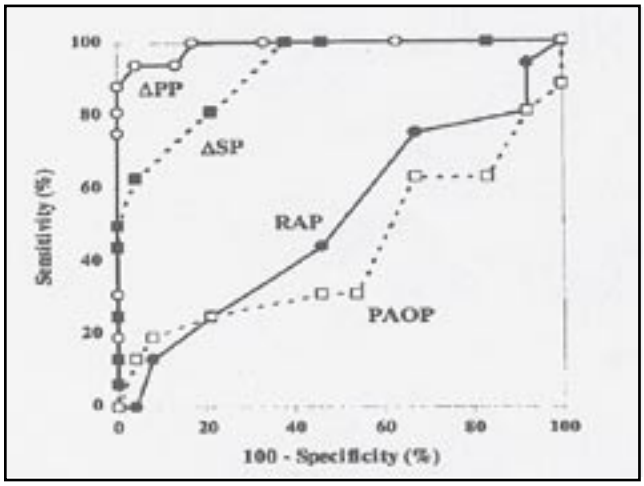

Figure 4.

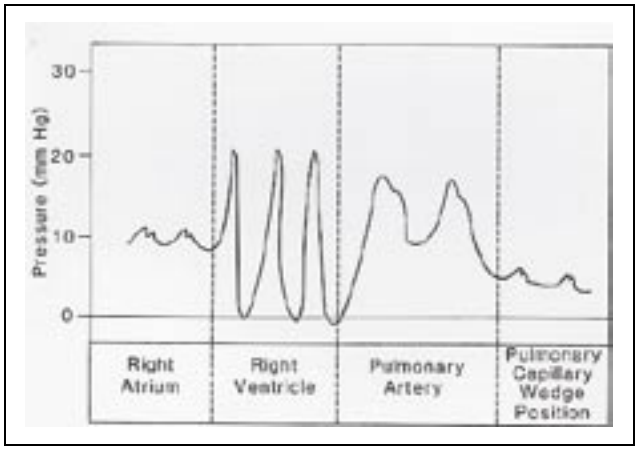

Figure 5.

Interpretation of Pressure Waveforms

Under most conditions, the waveforms obtained as the PA catheter (PAC) is advanced through the right atrium, right ventricle, and into the pulmonary artery to a wedged position are readily identified as characteristic of each segment of the circulation as it is traversed, as demonstrated in Figure 5. While waveform recognition is extremely helpful in positioning the catheter, and often makes the use of fluoroscopic techniques unnecessary, it is essential for the measurement and interpretation of waveforms displayed during PA catheterization to be correlated to the ECG tracing so that specific components of the waveform can be identified and various pitfalls in measurement of intravascular pressure can be avoided.

The Normal Pressure Waveform: In sinus rhythm, the atrial pressure waveform is characterized by two major positive deflections (A and V waves) and two negative deflections (X and Y descents) (Figure 6). A third positive wave, the C wave, is sometimes seen. The A wave results from atrial systolic contraction and is followed by the X descent as the atria relax following contraction. The C wave results from closure of the atrioventricular valves and interrupts the X descent. After the X descent, the V (ventricular) wave is generated by

Table 2. *Complications of Pulmonary Artery (PA) Catheterization*

I.	Complications related to central vein cannulation
II.	Complications related to insertion and use of the PA catheter
	A. Tachyarrhythmias
	B. Right bundle branch block
	C. Complete heart block (pre-existing left bundle branch block)
	D. Cardiac perforation
	E. Thrombosis and embolism
	F. Pulmonary infarction due to persistent wedging
	G. Catheter-related sepsis
	H. Pulmonary artery rupture
	I. Knotting of the catheter
	J. Endocarditis, bland and infective
	K. Pulmonic valve insufficiency
	L. Balloon fragmentation and embolization

passive filling of the atria during ventricular systole. Lastly, the Y descent reflects the reduction in atrial pressure as the atrioventricular valves open. In correlating these waveforms to the ECG, the first positive pressure wave to follow the P wave is the A wave. The right atrial A wave is usually seen at the beginning of the QRS complex, provided that atrioventricular conduction is normal. The peak of the right atrial V wave normally occurs simultaneously with the T wave of the ECG, provided that the Q-T interval is normal.

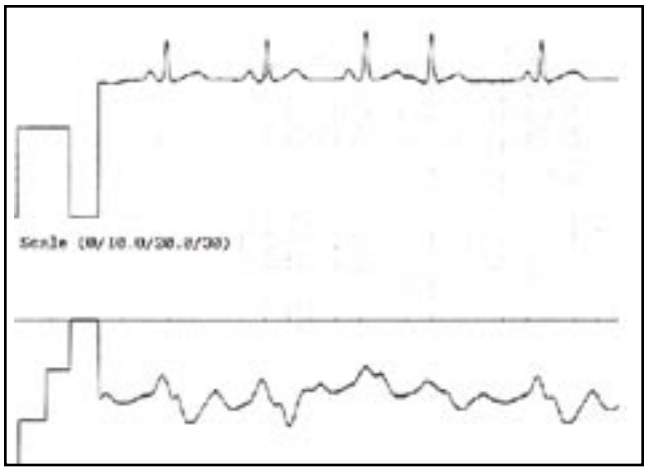

Figure 6.

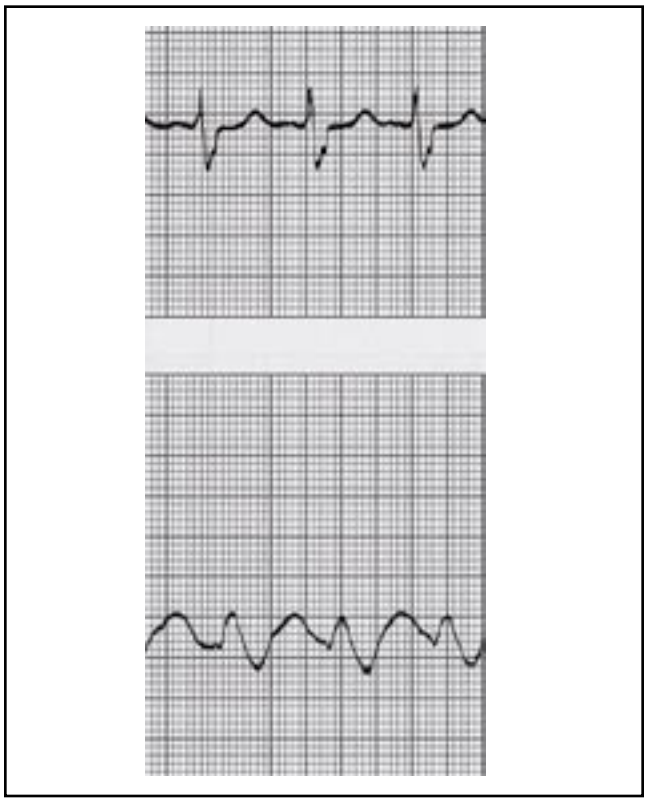

Figure 7.

The pulmonary artery waveform has a systolic pressure wave and a diastolic trough. A dicrotic notch due to closure of the pulmonic valve may be seen on the terminal portion of the systolic pressure wave. Like the right atrial V wave, the PA systolic wave typically coincides with the electrical T wave. The PA diastolic pressure (Ppad) is recorded as the pressure just before the beginning of the systolic pressure wave.

The Ppw tracing contains the same sequence of waves and descents as the right atrial tracing. However, when the atrial waveform is referenced to the ECG , the mechanical events arising in the left atrium (Ppw) will be seen later than those of the right atrium, because the left atrial pressure waves must travel back through the pulmonary vasculature and a longer length of catheter (Figure 7). Therefore, in the Ppw tracing the A wave usually appears after the QRS complex and the V wave is seen after the T wave. As such, the systolic pressure wave in the PA tracing *precedes* the V wave of the Ppw tracing. An appreciation of the latter relationship is critical when tracings are being analyzed to ensure that balloon inflation has resulted in a transition from an arterial (PA) to atrial (Ppw) waveform, and to detect the presence of a "giant" V wave in the Ppw tracing.

Common Problems Producing Erroneous Pressure Waveforms: Of the many problems causing artifact or erroneous tracings, the most commonly encountered are overdamping, catheter whip, overwedging, incomplete wedging, and Zone I catheter conditions.

Overdamping results from air bubbles within the catheter system or kinking, clotting, and fibrin deposition along the catheter course; many times these problems can be resolved by catheter flushing. The main effect of overdamping on the pressure waveform is to artifactually lower the systolic pressure and raise the diastolic pressure with consequent effects on interpretation (Figure 8).

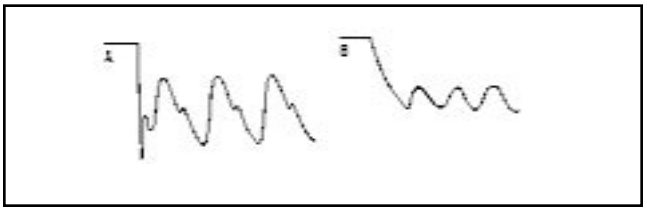

Figure 8. Rapid flush test: A) appropriately damped system; B) over damped system.

Catheter whip arises from cardiac contractions causing shock transients transmitted to the catheter. The results on the right ventricular or pulmonary arterial waveforms are an exaggerated diastolic pressure in some cycles, highlighting the need to avoid readings obtained by electronic systems.

Overwedging (Figure 9) is signaled by a rise in recorded pressure with balloon inflation as the balloon herniates over the catheter tip or the tip is pushed into the vessel wall with continued fluid ingress elevating the measured pressure. Overwedging requires repositioning of the catheter.

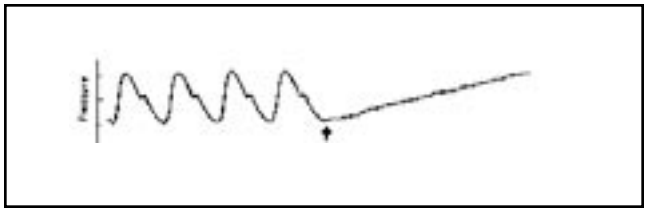

Figure 9.

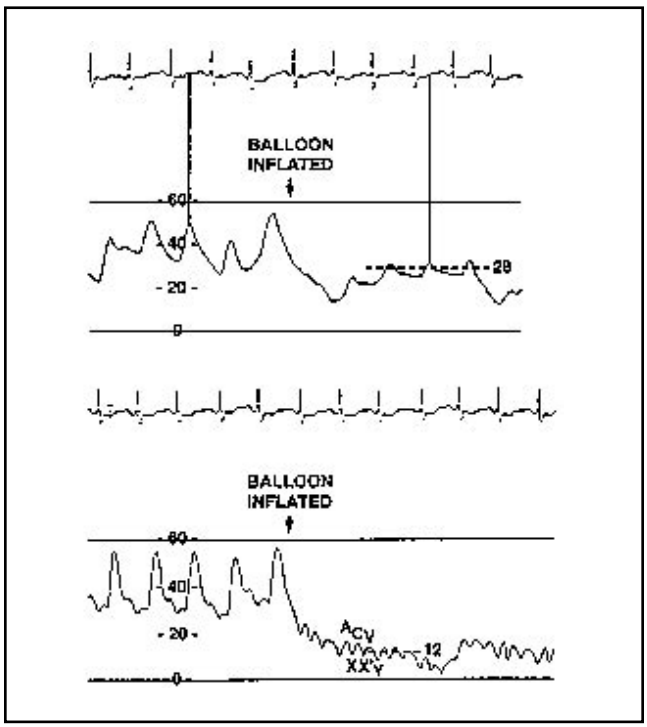

Figure 10. Incomplete wedge pressure (Ppw). Top: With balloon inflation, there is a decrease in pressure to a value that approximates pulmonary artery diastolic pressure (Ppad). The clinical setting (ARDS) is usually associated with a large Ppad-Ppw gradient. Review of the tracings indicates that there is a single positive wave coinciding with the electrocardiographic T wave after balloon inflation, a pattern inconsistent with a left atrial waveform. Bottom: Waveforms after the catheter had been retracted, the balloon inflated, and the catheter floated to a full wedge position. Now, there is a large Ppad-Ppw gradient and the tracing after balloon inflation is consistent with a left atrial waveform. The incomplete wedge tracing yielded an incorrect measurement of the wedge pressure as 28 mm Hg, substantially higher (in a very clinically relevant sense) than the true wedge pressure of approximately 12 mm Hg.

Incomplete wedging (Figure 10) and Zone I positioning of the catheter can be subtle but are important to identify since erroneous and often overestimation of Ppw occur.

Zone I conditions of the lung refer to those segments of the lung in which alveolar pressure exceeds pulmonary vascular pressure and hence there is no flow (Figure 11).

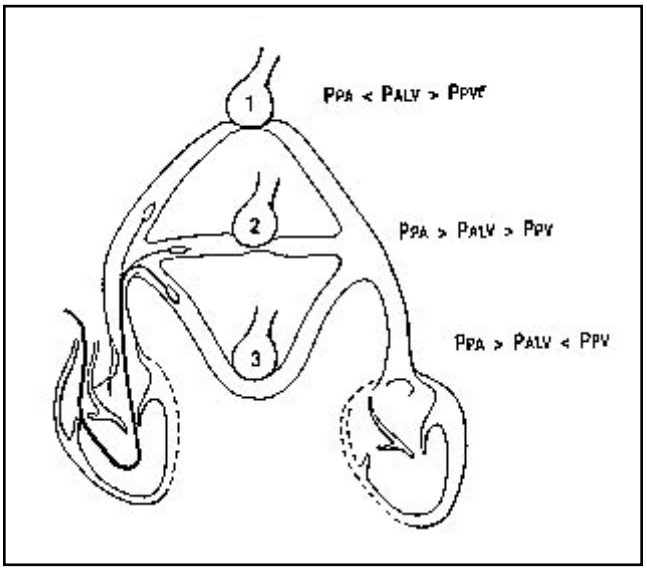

Figure 11. Lung zones.

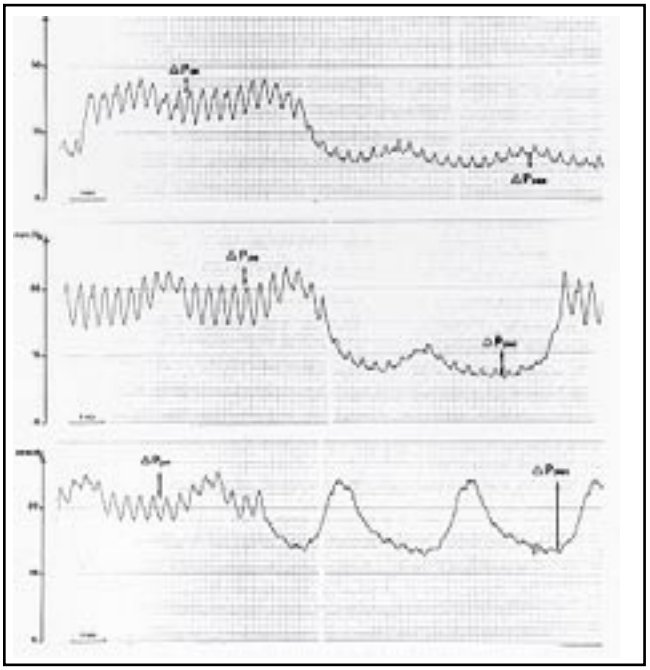

Figure 12. Pressure tracings recorded in the same patient at different levels of end-expiratory pressure-zero (ZEEP) on the top panel, 15 cm H_2O in the center panel, and 20 cm H_2O in the bottom panel.

This phenomenon is uncommon when the catheter is floated into position since this typically results in Zone II or III positioning. It would be more likely to result from forceful positioning of the catheter, hypovolemia emerging after placement, or with large increases in PEEP. This condition should be considered when changes in Ppw track PEEP changes exactly or when the excursion in pulmonary artery systolic pressures with respiration exceed those Ppw significantly (Figure 12).

The Correlation of Pressure to Ventricular Preload and Volume: The use of Ppw as a measure of left ventricular end-diastolic pressure and hence preload depends on the Ppw closely reflecting pulmonary venous, left atrial, and left ventricular pressures, that is, with minimal pressure gradient across the system. One potential confounder to interpretation of intravascular pressures is the fluctuation in intrathoracic pressure related to the respiratory cycle. The effect of varying intrathoracic pressure on the wedge (Ppw) pressure is seen in Figure 13. The top line is a Ppw tracing and the bottom in the intrapleural (Ppl) pressure. In this example the patient is receiving assisted ventilation. Arrows indicate end expiratory pressures. Negative deflections in Ppl and Ppw pressures result from inspiratory muscle activity, and subsequent positive deflections represent lung inflation by the ventilator. At end expiration, the respiratory system has returned to its relaxed state and Ppl is back to baseline (-2 cm H_2O). Transmural wedge pressure remains approximately constant throughout the ventilating cycle. Since Ppl is not usually measured clinically, it is necessary that Ppw be recorded at a point where Ppl can be reliably estimated (*ie*, end-exhalation, assuming no expiratory muscle activity).

The correlation of pressure to volume is further complicated by a variety of conditions that cause the ventricle to be effectively stiff (diastolic dysfunction or pericardial disease) or conditions that cause juxta-cardiac pressure to rise related to positive pressure ventilation (PEEP, intrinsic PEEP [PEEPi], active expiratory effort) (Figure 14).

The effects of PEEP in conditions such as ARDS are often blunted, since the stiff lungs of these patients do not distend greatly with high ventilator pressures and hence minimal increases in juxta-cardiac pressure are encountered. However, in cases in which PEEPi exists in chronic obstructive pulmonary disease (COPD) / asthma patients undergoing mechanical ventilation, or in agitated / obstructed patients with very active expiratory muscle effort, cardiovascular effects may be large. This effect is shown in Figure 15, where the increase in blood pressure and cardiac output despite a fall in wedge pressure and esophageal pressure is shown during a brief interruption in positive pressure ventilation in a patient with COPD.

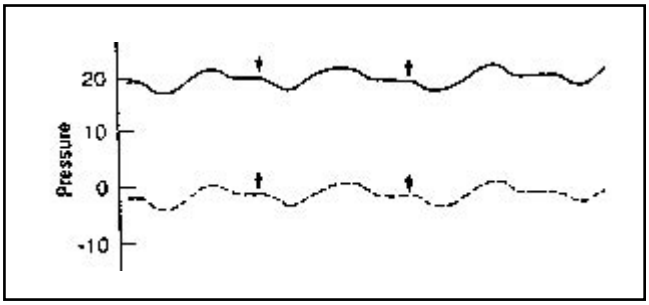

Figure 13.

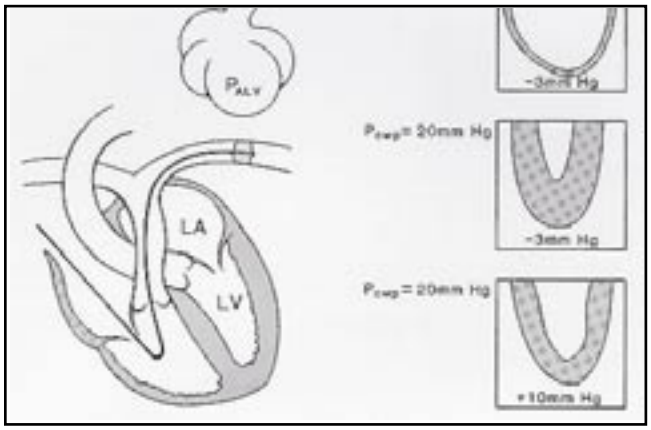

Figure 14.

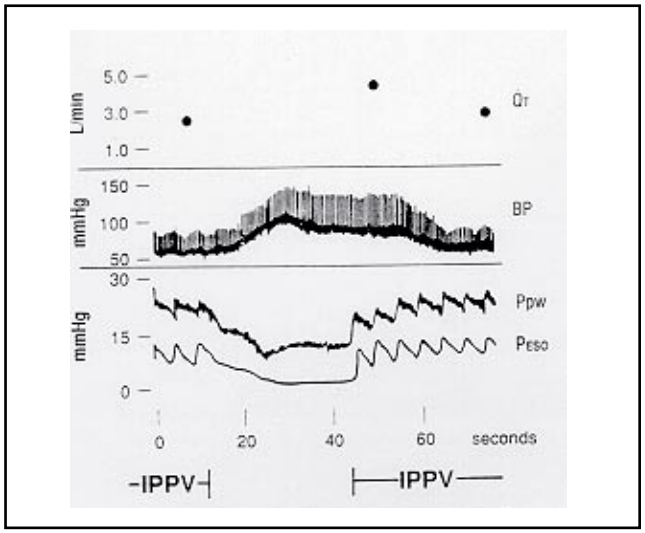

Figure 15.

This constellation of problems is best avoided by:

- Awareness of their existence
- Reading pressure tracings at end expiration
- Considering measures (sedation, ventilator adjustment, paralysis) that diminish or eliminate PEEPi
- Considering a ventilator disconnect in patients with severe airflow obstruction and PEEPi to demonstrate limitation to venous return
- Using a fluid challenge when effective "diastolic" dysfunction may be present, to determine "preload reserve"

In determining the response to a fluid challenge, it is necessary to note that a minimum of 500 mL of crystalloid is required and even then small effects on cardiac output and arterial blood pressure are typically seen. One study has suggested that the use of a drop in the right atrial pressure with respiration is a useful indicator of preload reserve.

Specific Disorders: Tricuspid regurgitation is encountered in conditions with direct valvular injury (*eg*, endocarditis) and generally in right heart failure. It is characterized by a prominent and broad V wave and a steep Y descent; the latter is often most useful for making this diagnosis (Figure 16). It is useful to note tricuspid regurgitation not only for its implications for underlying disorders but also because it will confound thermal dilution cardiac output determination.

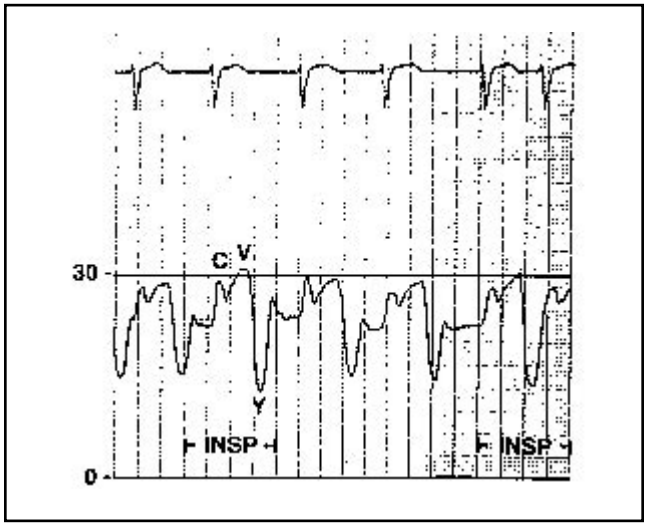

Figure 16. Giant V wave in right atrial waveform indicates tricuspid regurgitation.

Mitral regurgitation is characterized by a giant V wave that may confound distinction between the PA and Pwp tracings (Figure 17). Significant mitral regurgitation may be present without a giant V wave (ascribed to enlarged and compliant left atrium which does not exhibit a large pressure excursion with the additional volume) and a number of conditions can cause a giant V wave in the absence of mitral regurgitation (hypervolemia, VSD).

Right ventricular infarction is characterized by an elevated right ventricular end-disatolic pressure at initial passage of the catheter with narrow pulse pressures when there is hemodynamic compromise. This same pattern can also be present in conditions causing acute right heart failure secondary to increases in pulmonary vascular resistance (*eg*, pulmonary embolus) but in these latter conditions there will be a large PAD-Ppw gradient reflecting the increase in pulmonary vascular resistance.

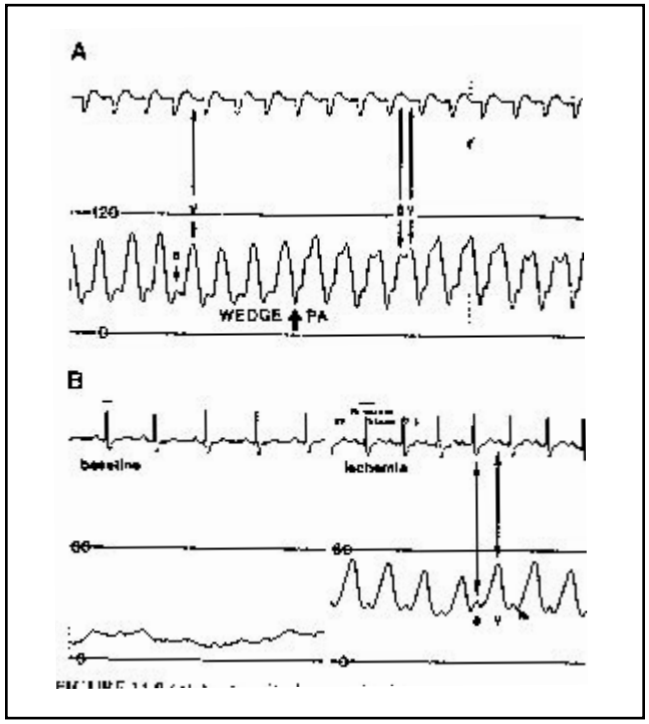

Figure 17. A) Acute mitral regurgitation with giant V wave in pulmonary wedge tracing. The pulmonary artery (PA) tracing has a characteristic bifid appearance due to both a PA systolic wave and the V wave. Note that the V wave occurs later in the cardiac cycle than the PA systolic wave, which is synchronous with the T wave of the electrocardiogram. B) Intermittent giant V wave due to ischemia of the papillary muscle. Wedge tracings are from same patient at baseline and during ischemia. Scale in mm Hg.

Interpretation of Flows and Parameters of Oxygen Delivery

In most clinical settings, cardiac output is determined by thermal dilution. In addition to a number of technical conditions making the measurement unreliable, tricuspid regurgitation may be present and cause underestimation (usually) or overestimation (rarely) of cardiac output. Under this circumstance, determination of cardiac output by Fick may be useful.

Determination of whether a measured flow is adequate is usually best judged by peripheral parameters of perfusion (*eg*, urine volume, presence of lactic acidosis) or by the mixed venous oxygen saturation ($Sv\overline{O}_2$). Low $Sv\overline{O}_2$ (< 60 %) strongly suggests inadequate oxygen delivery and anemia, hypoxemia, or inadequate cardiac output should be sought and corrected. Interpretation of a high $Sv\overline{O}_2$ in high-output states is difficult. Accordingly, the greatest utility of modified catheters which permit continuous monitoring of $Sv\overline{O}_2$ is in circumstances in which there is risk for it to be low and therapy can be directed at early recognition of this phenomenon (*eg*, postoperative cardiac surgery patients).

Echocardiography

Many of the problems of relating measured pressures to ventricular preload can be addressed by cardiac imaging by echo. In addition, this diagnostic tool is useful for identifying a host of structural abnormalities. It should be considered as an adjunct to pulmonary artery catheterization. As technology permits more continuous monitoring by transesophageal route, its use in the ICU is likely to expand.

Useful Applications of Echocardiography in the ICU

- Identification of ischemia
- Correlation of pressure to volume and identification of diastolic dysfunction
- Characterization of valve lesions, VSD, ASD
- Identification of pericardial disease
- Identification of right-left heart interactions in acute right heart failure

Selected Reading

Connors AF, Speroff T, Dawson NV, et al. The effectiveness of right heart catheterization in the initial care of critically ill patients. JAMA 1996; 276:889-897

Recent retrospective study employing case-matching methodology which suggested the pulmonary artery catheter (PAC) is associated with a poor outcome, perhaps apart from patient risk factors

Fuchs RM, Heuser RR, Yin FC, et al. Limitations of pulmonary wedge V waves in diagnosing mitral regurgitation. Am J Cardiol 1982; 49:849-854

Leatherman JW, Marini JJ. Clinical use of the pulmonary artery catheter. *In:* Principles of Critical Care, 2nd Ed. Hall JB, Schmidt GA, Wood LDH (Eds). New York: McGraw Hill, 1998; 155-177

Excellent, concise review of use of PAC

Magder S, Georgiadis G, Cheone T. Respiratory variations in right atrial pressure predict the response to fluid challenge. J Crit Care 1992; 7:76-85

Michard F, Boussat S, Chemla D, et al. Relation between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. Am J Respir Crit Care Med 2000; 162:134-138

Michard F, Teboul JL. Predicting fluid responsiveness in ICU patients. Chest 2002 121:2000-2008

Michard F, Teboul JL. Using heart-lung interactions to assess fluid responsiveness during mechanical ventilation. Crit Care 2000; 4:282-289

Perret C, Tagan D, Feihl F, et al. The pulmonary artery catheter in clinical care. Oxford, England: Blackwell Science, 1996; 347

Excellent monograph, includes case studies

Russell JA, Phang TP. The oxygen delivery / consumption controversy: Approaches to management of the critically ill. Am J Respir Crit Care Med 1994; 149:533-537

Shah KB, Rao TLK, Laughlin S, et al. A review of pulmonary artery catheterizations in 6,245 patients. Anesthesiology 1984; 61:271

Documents risks and complications

Teboul JL, Besbes M, Andrivet P, et al. A bedside index assessing the reliability of pulmonary artery occlusion pressure measurements during mechanical ventilation with positive end-expiratory pressure. J Crit Care Med 1992; 7:22-29

Notes