Hemodynamic Monitoring in the Intensive Care Unit

Hayan Al Maluli, MD and Christine M. DeStephan, MD

Abstract
Caring for critically ill patients has dramatically changed over the past two decades. Advancement in therapeutic modalities has been built on a foundation of a thorough knowledge of the hemodynamic principles applied in modern intensive care units (ICU’s). Mastery of hemodynamic monitoring tools in the critical care setting is essential to optimal patient care. Pulmonary artery catheters, invasive arterial pressure monitors, and central venous catheters are the three most utilized tools for this purpose. The scientific principles underlying their indications, their insertion techniques, and data on their efficacy are discussed here.

Keywords — Intensive care, pulmonary artery catheters, invasive arterial pressure monitors, cardiac output, central venous catheters

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The complete assessment of unstable patients often requires knowledge of their intravascular volume and pressure status. While indirect measurements and assumptions can often be made at the bedside, a more direct way of obtaining this information is frequently needed. Pulmonary artery catheters (PAC’s) are utilized to obtain left and right sided cardiac pressures. The accuracy of the information obtained depends on avoidance of the pitfalls and on meticulous insertion techniques. Invasive blood pressure monitoring is often required in both extremes of blood pressure value. Careful assessment of the arterial anatomy helps to prevent complications related to accessing the arteries. Central venous catheters serve as essential routes of administering resuscitation medications and fluids. Furthermore, they provide invaluable hemodynamic information that is readily usable.

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I. PULMONARY ARTERY CATHETERS

BACKGROUND
Before 1970, the study of the central circulation in man consisted of right and left heart catheterization with semirigid catheters. Catheter placement was performed exclusively in the catheterization laboratory under fluoroscopy and required highly specialized training. Employing the principle of a flow directed balloon-tipped catheters used in dogs by Lategola and Rahn in the early 50’s, Swan and Ganz developed a flexible catheter that uses blood flow to guide its direction using a balloon at its tip as the mainsail. These catheters were easy to use, had a high rate of success in reaching the pulmonary artery in a short time, and had a significantly lower complication rate (thrombosis, arrhythmia provocation, etc.). In addition, the new catheters were inserted at the bedside without need for fluoroscopy.1

INDICATIONS
Since its advent four decades ago, the pulmonary artery catheter (PAC) has undergone significant additions. Modern PACs allow for pressure monitoring either intermittent or continuous, measurement of cardiac output (CO), fluid and pressor infusion, even cardiac pacing. Therefore, it is no surprise that the indications for its use are many and various. Here we describe the most common indications in some detail and then briefly cite less common ones.

1- Evaluation of pulmonary edema: The differentiation between cardiogenic and non-cardiogenic pulmonary edema relies primarily on one parameter, which is pressure in the pulmonary circulation. If the pressure in the pulmonary circulation, specifically pulmonary artery occlusion pressure (PAOP), is elevated then the lung edema is likely secondary to either systolic or diastolic cardiac dysfunction. On the other hand, the presence of alveolar edema with a PAOP < 18 mmHg is one of the diagnostic criteria for Adult Respiratory Distress Syndrome (ARDS).2 The PAC allows for an accurate estimation of the PAOP and therefore is considered superior to clinical assessment alone in guiding the management of pulmonary edema in critically ill patients.3

2- Diagnosis and management of shock: The three most common causes of shock are: hypovolemic, cardiac, and septic.
While history and physical examination may reveal the majority of causes of hypovolemic shock including bleeding and profound dehydration among others, the differentiation between cardiogenic and septic shock is occasionally perplexing as predisposing conditions often coexist in the same patient. PACs provide invaluable information that may establish the diagnosis in most cases. Measurement of cardiac output (CO) and systemic vascular resistance (SVR) often yield the data necessary to make a correct guess. In the hypotensive patient (shock), a high CO in the presence of low SVR is often indicative of sepsis, especially if the patient had already received fluids. Normal or even elevated mixed venous blood oxygen saturation (SvO₂) is classically present in septic patients. On the other hand, a low CO with a high SVR, driven by compensatory sympathetic nervous system, is almost pathognomonic for cardiogenic shock. A low SvO₂ in this case represents a more avid extraction of oxygen by the peripheral tissues to compensate for diminished blood flow via decreased cardiac output. PAOP is the parameter that helps the most in differentiating hypovolemic from cardiogenic shock, low in the former and high in the latter. Obtaining the PAC can guide management as well as direct clinicians toward the correct diagnosis. Importantly, a patient’s response to inotropic or vasopressor support, either intermittent or continuous, can be monitored via quantifiable hemodynamic parameters. Last, the catheters can serve as an intravenous access for the administration of medications and resuscitation fluids concomitantly since modern catheters contain multiple ports.

3- **Unexplained pulmonary hypertension:** Pulmonary artery catheters can directly measure the pulmonary arterial pressure (pre-capillary pressure) and compare it to an accurate estimate of the PAOP (post capillary pressure). A diastolic pulmonary arterial pressure that is higher than the PAOP suggests a non-cardiogenic (i.e. post- capillary) source of the pulmonary hypertension. Currently, invasive testing modalities are the only option for accurately diagnosing and differentiating the types of pulmonary hypertension.

4- **Other indications:** summarized in Table 1.

### CONTRAINDICATIONS

The 1998 American College of Cardiology Consensus Document indicated relative and absolute contraindications to the placement of pulmonary artery catheters. All medical decisions require educated clinical judgment. The contraindications are summarized in table 2.

### OUTCOMES

Despite many indications for the pulmonary artery catheters, multiple trials have been unable to prove a mortality benefit and thereby justify routine use in certain clinical conditions. Indications for use include, but are not limited to:

1. **Management of shock:** Clinical trials compared the use PAC to no PAC or to use of central venous pressure catheters (CVP) in the guidance of management of shock. Those trials failed to show improved survival.
2. **ARDS:** By the same token, PAC failed to improve outcome when compared to CVP catheters in patients with ARDS.
3. **Heart failure:** In the Evaluation Study of Congestive heart failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial, the addition of PAC to clinical management protocols resulted in more adverse events without any beneficial effect on mortality.

Subsequently, an expected decline in the use of pulmonary artery catheters was noticed. Significant debate still exists regarding the validity of those studies in evaluating the value of PAC use in various clinical settings. The main argument in favor of routine PAC use holds that it is a diagnostic modality that pulmonary artery catheters have revealed much about hemodynamics and their interaction with disease states.

### Equipment

The original catheter used by Swan and Ganz was a 5 French (1.7 mm) caliber dual lumen catheter that had an inflatable balloon at the tip.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Findings</th>
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<tbody>
<tr>
<td>Acute MI with shock:</td>
<td>Elevated RA pressure with normal PAOP.</td>
</tr>
<tr>
<td>1. RV infarction</td>
<td>&gt;7% O2 sat difference between RA and PA</td>
</tr>
<tr>
<td>2. VSD</td>
<td>Giant v waves on the PA wedge tracing.</td>
</tr>
<tr>
<td>3. Acute MR</td>
<td>Equalization of RA pressure and PAOP with blunted y descent</td>
</tr>
<tr>
<td>4. Pericardial tamponade</td>
<td>Equalization of RA pressure and PAOP with deep x and y descent</td>
</tr>
</tbody>
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**Constrictive Pericarditis**

**Chronic Heart Failure:**

1. **Pseudosepsis:** due to excessive vasodilation.
2. **Cardiac amyloidosis:**

Table 1: Other indications for pulmonary artery catheter. RV: Right ventricle. VSD: Ventricular septal defect. MR: Mitral Regurgitation. RA: Right atrium. PAOP: Pulmonary artery occlusion pressure. PA: Pulmonary artery. CO: Cardiac output. SVR: Systemic vascular resistance.
Modern PAC’s are 110 cm in length, up to 7 French (2.3 mm) in diameter, catheters that can have as many as five ports. All PAC’s share the same basic components of a balloon at the tip that is typically inflated with 1.5 cc of air, a thermistor located 4 cm proximal to the tip, and a right atrial lumen that ends with an opening about 30 cm from the tip and opens into the right atrium. A thermistor, which is a hybrid of thermal and resistor, is a non-metal, usually ceramic device that measure of resistance that varies with temperature more than other types of resistors. Therefore it can be used to sense temperature.

Several additions to the PAC enhance its utility. A thermal filament that generates heat pulses and allows continuous measurement of the cardiac output is available in some catheters (discussed later). Another optional feature is a fiber-optic system that can constantly measure the mixed venous oxygen saturation. Some catheters have an additional port with an opening that is 14 cm proximal to the tip, which is used to thread transvenous pacers. 

### Table 2: Relative and absolute contraindications for the placement of pulmonary artery catheter. ICD: Implantable cardioverter defibrillator. PAC: Pulmonary artery catheter.

<table>
<thead>
<tr>
<th>Relative Contraindications</th>
<th>Absolute Contraindications</th>
</tr>
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<tbody>
<tr>
<td>- Coagulopathy.</td>
<td>- Right sided endocarditis.</td>
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<tr>
<td>- Recent implantation of a pacemaker or ICD: in this case placement of PAC under fluoroscopy is recommended.</td>
<td>- Mechanical tricuspid or pulmonic valves.</td>
</tr>
<tr>
<td>- Left Bundle Branch Block: Temporary transvenous pacer should be placed prior to attempting PAC placement.</td>
<td>- Right heart thrombus or tumor.</td>
</tr>
<tr>
<td>- Bioprosthetic tricuspid or pulmonic valves.</td>
<td>- Terminal illness in which aggressive treatment is considered futile.</td>
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Pulmonary artery catheter insertion technique

After establishing a venous access usually in the internal jugular (IJ) or subclavian vein (SC), a large bore introducer catheter is placed first. The PAC is then inserted into the vein through the introducer. A pressure sensor at the tip of the catheter detects pressure changes as the catheter is being advanced. Initially, a venous wave form is noticed as fine baseline oscillations. These oscillations reflect the superior vena cava (SVC) or right atrial pressure variations throughout the cardiac circle. Note that a detailed illustration of the various waves may be hard to see on the monitor due to the miniature scale.

Once oscillations are observed, the balloon is inflated with 1.5 cm of air and the catheter pushed in gently to allow the blood flow to guide it into the right ventricle (RV) and pulmonary artery (PA). Entry into the RV is heralded by a pulsatile wave form that reflects the contractile nature of the RV. The diastolic pressure of the RV is typically equal to the RA pressure (1-6 mmHg) in the absence of tricuspid valve stenosis, whereas the systolic pressure is usually in the range of 15-30 mmHg. Further advancement of the catheter allows passage through the pulmonic valve into the PA. The pressure wave in the pulmonary artery is notable for a step-up in the diastolic pressure and preservation of the systolic pressure of the RV. The step-up in the diastolic pressure is a result of the resistance that the pulmonary circulation exhibits on blood flow. The diastolic PA pressure is in the range of 6-12 mmHg. An arterial dicrotic notch, coincident with pulmonic valve closure, also helps in differentiating the PA pressure wave form.

Once the catheter tip is about 10-15 cm past the RV or 45-55 cm from the skin when right internal jugular vein is used, a disappearance in the pulsatile PA pressure wave form is usually detected. This means that the balloon has occluded one of the lobar pulmonary arteries that is “wedged” it. An oscillatory baseline is again noted but this time at a slightly higher pressure that reflects the left atrial (LA) or left ventricular diastolic pressure (LVEDP). This pressure is usually equal to the pulmonary artery diastolic pressure and is one of the most important measurements obtained from the PAC. The wedge pressure, also termed pulmonary artery occlusion pressure (PAOP), is recorded before the balloon is deflated to prevent injury to the PA. Likewise the syringe used to inflate the balloon should be disconnected from the PA port to prevent accidental inflations. A chest x-ray should always be obtained to confirm location and screen for placement complications (e.g. pneumothorax).

In addition to the universal protocols followed during any central venous access establishment, the PAC placement procedure requires specific monitoring. Because the catheter is passing through the heart and pulmonary artery, a higher risk of bleeding from damage to those structures warrants frequent blood pressure checks. More importantly, continuous rhythm monitoring is mandatory as tachy- or brady-arrhythmias may occur when the PAC tip touches the endocardium. Continuous pulse oximetry is required.

TROUBLESHOOTING

Inability to pass the catheter into the RV can sometimes happen especially if there is significant tricuspid valve regurgitation (TR). Placing the catheter by temporarily filling the balloon with sterile saline and placing the patient in left lateral position is sometimes effective. The saline should be
replaced with air once the catheter is in the RV. If this maneuver does not work, then placement under fluoroscopy in the catheterization lab should be pursued.

Sometimes there is difficulty in passing the catheter into the pulmonary artery, especially in cases of pulmonary hypertension. Having the patient inhale to increase the right ventricular blood flow can be helpful. Infusing cold saline into the catheter can make it stiffer and allow for better maneuvering. Stiffer catheters may be used in such cases with an increased risk of vascular or cardiac rupture. Failure to advance the catheter safely is an indication for fluoroscopic placement.

COMPLICATIONS

Several sources of complications exist with the use of PAC’s. Those complications, which are potentially avoidable, have negatively influenced the outcome of patients receiving PAC as part of their routine management protocols and therefore limited the widespread use of those catheters. Some authors attribute the lack of mortality benefit with the use of pulmonary artery catheters to the higher risk of complications from the PAC especially in patients who are less sick.

1) RELATED TO CATHETER INSERTION:

Injury to the lung apex can result in pneumothorax, the overall risk of which is estimated between 1-3%. Several factors make this complication more likely and should be actively avoided whenever possible. Multiple attempts at obtaining the venous access, especially by an inexperienced operator, carry higher risk. Anatomically difficult patients, either obese or very thin, can also impose challenges, hence higher risk. Emergent insertions, especially when not guided by ultrasound can result a higher rate of all complications. Chest x-rays must be obtained immediately after PAC placement or with any suspicion of respiratory or circulatory distress in order to allow early detection and management of this serious complication.

Bleeding can occur as a result of coagulopathy, arterial puncture, or both. Correction of platelets disorders or abnormal coagulation studies prior to the procedure should always be considered when appropriate. The subclavian approach is best avoided if coagulopathy is not corrected because the clavicle prevents adequate compression of the vessels in case of a bleed. Damage to the artery can sometimes occur causing large hematomas or signs of arterial insufficiency. The carotid in IJ insertions or less often, the subclavian in SC vein insertions are particularly susceptible. Adequate pressure over the carotid will treat most cases. On rare occasions, cannulation of the carotid artery can lead to neurologic complications. Vascular surgery is best consulted in such cases to ensure adequate closure of the arterial lesions.

Cardiac arrhythmias are common. They range from simple premature contractions to serious ventricular tachycardias or heart blocks and therefore the exact incidence of occurrence is variable. Nevertheless, life threatening arrhythmias are definitely uncommon. Irritation of the right atrium or ventricle can precipitate tachyarrhythmias originating in either of them. Injury to the AV node can lead to transient or even permanent heart blocks. The risk of causing complete heart block in cases of preexisting left bundle branch block (LBBB) was a former contraindication to the placement of PAC in the absence of a temporary transvenous pacemaker. However, this complication is rare therefore a transvenous pacer is not warranted prophylactically. Placing transcutaneous pacer pads on the chest and having the equipment for temporary transvenous pacing at the bedside should suffice.

2) AFTER PLACEMENT:

Thrombosis of the catheter can manifest clinically by pulmonary emboli, internal jugular vein thrombosis, and superior vena cava syndrome. However, these clinical complications are very rare with an incidence of 0.1-6%. More commonly the thrombosis of the PAC is detected by a malfunction such as inability to draw blood from the catheter or poor PA wave form. In those cases the catheters should be removed and anticoagulation started as indicated clinically.

Infection of the catheter site or the catheter itself can be best avoided by adhering to sterile placement techniques (cleansing the skin, full barrier precautions among others). Additionally, antibiotic-coated catheters can reduce risk. Routine daily assessment of the need for the catheter allows early discontinuation and helps in preventing infections. Serious infections are rare, and sepsis occurs in 1-3% of all patients. Staphylococci, both coagulase negative and positive, are the most commonly cultured pathogens.

The most dreaded complication is pulmonary artery rupture. Fortunately, this is a very rare complication (<1%) however the mortality rate is very high (~50%). Rupture can result from inflating the balloon in a small vessel or from over inflating in a large one. Withdrawal of the catheter when the balloon is inflated is risky and may lead to PA rupture. Occasionally the rupture can occur from the catheter tip during insertion although this is unlikely since the balloon is usually located at the very tip of the catheter. In cases of PA rupture, hemodynamic instability ensues and the patient usually complains of hemoptysis. Occasionally the rupture can be subtle and presents as a new infiltrate on the chest x-ray or as a hemothorax. PA angiography is recommended in stable patients and interventional radiological embolization of
the vessel can stop the bleeding. In unstable patients surgical options should be explored immediately.

II. MEASUREMENT OF CARDIAC OUTPUT

Cardiac output (CO) is an important parameter not only because it reflects cardiac function but also because it is an indicator of circulatory sufficiency since it, along with systemic vascular resistance (SVR), is a major driver of blood pressure (BP= CO x SVR). The pulmonary artery catheter allows for the measurement of the cardiac output in two different ways.

FICK METHOD

Principle:

The Fick principle relies on the observation that the total uptake of a substance by the peripheral tissues is equal to the product of the blood flow to the peripheral tissues and the difference in this substance’s content between arterial and venous blood.

Method:

Using an indicator substance, the Fick method allows for the calculation of the cardiac output given the availability of the following information:

1- The amount of substance uptake by the peripheral tissue.

2- The amount (content) of this substance in the blood supplying this tissue (i.e. arterial blood).

3- The amount of this substance in the blood leaving this tissue (i.e. venous blood).

An excellent predictor substance is oxygen because oxygen uptake and blood content can be measured relatively easily.

Using the following abbreviations in applying the previous principle:

VO₂ = oxygen uptake by tissues

CaO₂ = Arterial content of oxygen

CvO₂ = Venous content of oxygen

These variables yield the following formula:

\[ \text{VO}_2 = (\text{CO} \times \text{CaO}_2) - (\text{CO} \times \text{CvO}_2) \]

Which means that:

\[ \text{CO} = \frac{\text{VO}_2}{(\text{CaO}_2 - \text{CvO}_2)} \] (1)

Using a gas analyzer, VO₂ can be directly measured by comparing the difference between the oxygen content in inspired and expired air. More practically, and conveniently, VO₂ can also be assumed based on body weight. VO₂ of 3 ml/min/kg or 125 ml/min/m² for average individuals is used in most units.

CaO₂ and CvO₂ can be calculated using the following formulas:

\[ \text{CaO}_2 = 1.36 \times \text{Hb} \times \text{SaO}_2 \] (2)

\[ \text{CvO}_2 = 1.36 \times \text{Hb} \times \text{SvO}_2 \] (3)

Where SaO₂ is arterial oxygen saturation, SvO₂ mixed venous oxygen saturation (pulmonary artery blood), and Hb is hemoglobin concentration. The constant 1.36 represents the amount of oxygen that each molecule of Hb can carry when fully saturated.

Integrating formulas (2) and (3) into (1) yields the following formula that allows for an easy calculation of the CO:

\[ \text{CO} = \frac{\text{VO}_2}{1.36 \times \text{Hb} \times (\text{SaO}_2 - \text{SvO}_2)} \] (4)

Pitfalls:

There are three major potential sources of error with this formula and its application:

First: this formula should reflect total body consumption of oxygen (VO₂) but because the SvO₂ is actually sampled from the pulmonary artery, the lung tissue oxygen consumption is neglected. This is usually acceptable since the lungs normally consume less than 5% of whole-body O₂. In instances where there is inflammation in the lungs, such as ARDS or pneumonia among others, the contribution of the lungs to VO₂ can be as high as 20%. This means that in the presence of such inflammation in the lungs, the measured VO₂ will overestimate VO₂ by as much as 20%.

Second: in the presence of left to right intracardiac shunt, the SvO₂ will be falsely elevated.

Third: normally oxygen dissolved in the blood that is not carried by hemoglobin does not contribute significantly to the total amount of oxygen in the blood; hence formulas (2 and 3) are usually valid. However, at high fraction of inspired oxygen (FiO₂) the contribution of the dissolved O₂ to the total O₂ content can be underestimated.
Fourth: a great deal of error can occur with variations of body oxygen consumption, and assuming VO₂ may underestimate true consumption in hypermetabolic states and overestimate it in hypometabolic conditions.

THERMODILUTION METHOD

Principle:

If an indicator substance is added to the circulating blood, the rate of blood flow is proportional to the change in concentration of the indicator over time.

Method:

In this method, the indicator is a cold fluid which is injected into the right atrium via the right atrial port of the PAC. This cold fluid is then ejected by the right ventricle in an amount proportionate to the cardiac output (pulmonary flow). A thermistor located at the tip of the PAC measures the changes in temperature and transmits them to a computer system that calculates the cardiac output based on the changes in the temperature in a certain amount of time. Ideally, 10 cc of solution is injected. This solution used to be ice cold dextrose in water but more recently it was found that solutions at room temperature can yield reliable results as well.

Pitfalls:

First: In the presence of intracardiac left to right cardiac shunt, the shunted blood dilutes the injectate and warms the blood quickly thereby overestimating the cardiac output. Also, in the presence of intracardiac right to left shunt, a portion of the injectate is lost to the left side and does not reach the pulmonary artery, which leads to a more rapid dilution of the smaller amount of injectate hence an overestimation of the cardiac output.

Second: In the presence of tricuspid valve regurgitation, the cold injectate is recycled which leads to prolonged exposure of the catheter tip to the cold solution, falsely indicating a low cardiac output.

Third: in low output states the blood remains in contact with the myocardium for longer periods of time and is warmed by this contact. This would underestimate the true cardiac output since less cold blood arrives at the catheter tip.

Continuous cardiac output:

Technological advances of the PAC allow for a continuous measurement of the CO using the thermodilution method. The (Baxter Edwards Critical Care, Irvine, CA) catheter contains a 10 cm heat generating filament located 20-25 cm proximal to the thermistor-containing catheter tip. The heat generated by the filament replaces the injectate in changing blood temperature. Similar to the previous method, the change in temperature with time is used to calculate the cardiac output, however, this catheter records CO over 3 minutes, which provides more reliable information in the critically ill patient.

Pressures and waveforms

1) Pressures

Right atrial pressure

In normal individuals, the right atrial pressure ranges from 2 to 8 mmHg. This pressure is equal to the pressure in the superior vena cava (SVC) and collectively they are named the central venous pressure (CVP). The CVP is an indicator of intravascular volume status and is typically considered adequate at 10 mmHg. Respiratory movements transmit pressure changes to the compliant central venous system causing a normal drop in the CVP with inspiration; in cases of saturated venous capacity from adequate intravascular volume, those respiratory variations are blunted. The lack of respiratory variations of the CVP is a predictor of lack of response of the cardiac output to fluid infusions.

Normally, the right atrial pressure is smaller than the left atrial (LA) and PA pressures. In cases of left heart failure, the LA pressure is increased out of proportion to the RA pressure. In contrast, in pulmonary hypertension, the RA pressure might be higher than the left atrial pressure. The right and left atrial pressures are equal in cases of cardiac tamponade.

Pulmonary artery pressure

The PA pressure can be directly measured by the pulmonary artery catheter (PAC). The systolic pulmonary pressure is 15-30 mmHg and the diastolic is 4-12 mmHg. Typically, there is a small pressure difference between the diastolic PA pressure and the PAOP that is reflective of the LA pressure. In cases of left heart induced pulmonary hypertension, the PAOP is increased as much as the PA pressure. In cases of pure pulmonary arterial hypertension, the pressure gradient between the PA diastolic pressure and PAOP is increased. Therefore, as mentioned earlier, the PAC is an invaluable tool in the evaluation of pulmonary hypertension.

Pulmonary artery occlusion pressure (Wedge pressure):

The pulmonary artery occlusion pressure (PAOP), also known as wedge pressure, is one of the most important parameters.
obtained from the pulmonary artery catheter (PAC). As mentioned under insertion technique, the PAOP is obtained when the floated balloon stops at one of the branches of the pulmonary artery creating a static column of blood that extends from the tip of the catheter to the left atrium (LA). Since there is no blood flow (Q) between the catheter tip and the LA, the pressure difference between them is zero. This is derived from adjusting the following equation:

\[ Q = \frac{\Delta P}{R} \] (5)

Where

Q: is blood flow between the catheter tip and the LA.

\(\Delta P\): is the pressure difference between the catheter tip (PAOP) and LA (LAP).

R: the pulmonary vascular resistance between the catheter tip and LA.

By readjusting the equations we get:

\[ \Delta P = Q \times R \] (6)

There is no flow when the balloon is inflated (Q = 0), which means that \(\Delta P\) is equal to zero and that PAOP (the pressure at the catheter tip) is equal to LA pressure.

Under normal conditions, the LA pressure is equal to left ventricular end-diastolic pressure (LVEDP). Therefore, the PAOP can be used as a measurement of LVEDP. This allows the physician to predict the pressure in the left heart chambers without the risk involved in instrumenting them. Theoretically, the LVEDP and left ventricular end diastolic volume (LVEDV) or preload should always correlate. As we will see later, this simplistic assumption can lead to poor clinical decisions.

Furthermore, since the column of blood that is generated from inflating the balloon extends from the pulmonary arterial blood and includes the pulmonary venous circulation, the PAOP can be used as a surrogate of pulmonary circulation hydrostatic pressure, which is the pressure that determines, in addition to the pulmonary blood oncotic pressure, the movement of fluid between the vascular bed and the extracellular space in the lungs. This means that PAOP can be useful in evaluating pulmonary edema since it differentiates, ideally, between high hydrostatic pressure pulmonary edema and edema that results from increased vascular permeability such as in ARDS.

**Caveats**

Although the pulmonary artery catheter affords advantages in obtaining diagnostic data as described above, the technique must be employed with caution when guiding clinical management decisions. Several limitations are reviewed here:

1- Inaccurate zero pressure reference:

As with all measurements, a reference value needs to be established in order for the measured values to be valid. In the case of PAC obtained pressures, a zero reference value needs to be obtained prior to measuring pressures in any chamber. This is obtained by leveling the pressure transducer at the level of the atria which is marked externally by the intersection point between the fourth intercostal space and the right mid axillary line. This is called the phlebostatic axis, and it is valid only in the supine position. It is extremely important to position the transducer properly at the phlebostatic axis since a deviation of as little as 10 cm can cause an opposite change in the PAOP of 7.5 mmHg, which is clinically significant.

2- Catheter tip position

The pulmonary artery catheter measures the highest pressure surrounding its tip. In areas of the lung where the alveolar pressure may exceed the pulmonary venous pressure, the PAOP might actually reflect the alveolar pressure rather than the pulmonary venous pressure and therefore give a misleading estimate of the filling pressures of the left heart. This happens when the catheter tip is placed in the upper two thirds of the lungs, zones I and II, where the alveolar pressure is high and pulmonary venous pressure is low because blood is pooled down by gravity, or if the alveolar pressure is abnormally high such as with positive end expiratory pressure ventilation (PEEP) whether generated by the patient himself (auto or intrinsic PEEP), or intentionally induced by a ventilator (extrinsic PEEP) for example in cases of ARDS. Fortunately, the blood flow-guided catheters usually automatically follow the blood to the best perfused areas making this less of an issue than it may seem.

3- Problems with estimation of left ventricular end diastolic pressure.

Normally the LA pressure is equal to the LVEDP. However, in two common valvular abnormalities this may no longer be true. In mitral stenosis, the LA pressure is high despite the normal LVEDP. This means that the PAOP will overestimate the LVEDP. In contrast, in cases of aortic insufficiency, the regurgitant jet closes the anterior mitral leaflet prematurely while the left ventricle continues to fill from retrograde flow; this causes the PAOP to falsely underestimate the true LVEDP.
Problems with estimation of left ventricular end diastolic volume (preload).

The LVEDP is a factor of two important variables. It is directly related to LVEDV and inversely related to the compliance of the LV, which is the change in volume in response to change in pressure. In cases of poor LV compliance, a small change in volume can lead to an exaggerated increase in the pressure. This means that the pressure (LVEDP) is an invalid estimate of the volume (preload) in cases of poor ventricular compliance and is exemplified by left ventricular hypertrophy and restrictive cardiomyopathy.

Differentiating the pathogenesis of pulmonary edema.

There are two basic mechanisms of generating pulmonary edema. One is via elevated pulmonary capillary hydrostatic pressure and the other is increased permeability of the capillaries to fluid.

As mentioned under indications for PAC placement, the PAC may allow differentiation between these two mechanisms by providing the PAOP as a surrogate of the pulmonary capillary hydrostatic pressure. When the blood flow is absent, i.e. upon inflating the balloon, the pressures downstream from the catheter tip to the LA are equal (as explained by the equation 6 above). In addition, the true hydrostatic pressure that drives the pulmonary edema is the dynamic pressure, meaning the pressure of blood flowing in the capillaries. Normally, the dynamic pressure is very close to the static pressure due to the low resistance of the pulmonary veins. In cases of pulmonary venous constriction, as occurs with hypoxemia for example, the resistance to flow is high and therefore the wedge pressure, which is the post-venous or LA pressure, might be significantly lower than the capillary hydrostatic pressure (pre-venous pressure).

II) Wave forms

In addition to measuring pressures, the PAC provides graphic representations of the hemodynamics and these can be used to make the diagnoses of several cardiovascular abnormalities.

Right atrial wave form:

The normal right and left atrial wave form is composed of several positive and negative deflections (Fig 1).
Fig 2: Upper tracing is from the right ventricle (RV) and the lower tracing is from the pulmonary artery (PA). Note that there are two ways to differentiate between the two waveforms to locate the catheter tip without fluoroscopy. First, although the systolic pressures are the same in the absence of pulmonary valve stenosis, the PA diastolic pressure is higher than that of the RV (related to the PA resistance to blood flow). Second, in the absence of severe pulmonic valve regurgitation, there should be a notch, called the dicrotic notch, on the downslope of the PA tracing.

Pulmonary artery wedge or left atrial wave form:

The wedge pressure or the PAOP has basically the LA wave form configuration. This is similar to the right atrial pressure wave form except that the pressures in the left side are usually slightly higher (Fig 3).

Similarly, a prominent left sided v wave is seen with mitral regurgitation especially if acute, most notably with rupture of ischemic papillary muscle.

In cases of cardiac tamponade, the pressure in both the right and left atria is equal to the pericardial pressure. This is called equalization of pressures and is also seen in constrictive pericarditis. In tamponade the x descent is preserved but the y descent is blunted. In contrast, in constrictive pericarditis, the waves are not blunted, moreover, the x and y waves are deeper due to the initial higher pressure of the entire atrial tracing. Restrictive cardiomyopathy shares the same effect on the x and y waves as constrictive pericarditis, however, there is no equalization of pressures in this case.

Fig 3: Pulmonary artery occlusion pressure (PAOP) waveform. Notice that in the case of this patient the PAOP is around 5 mmHg which is normal. The fact that the pulmonary arterial pressure is elevated (Fig 2 lower tracing) in the absence of left sided congestion points towards an intrinsic pulmonary vascular process (i.e. pre-capillary pulmonary hypertension).

Wave form analysis artifact:

Non-pathological states can cause artifact in the wave forms which can lead to misinterpretation of the pressures.

A- Overdamping: disappearance of the pulsatile PA pressure wave-form can sometimes be the result of a clogged catheter rather than adequate inflation of the balloon and wedging. This can be due to air bubbles or clots and can be diagnosed by attempting to flush the catheter and observing for a rapid decrease and overshoot in the wedge pressure right after stopping the flush.

B- Whip artifact: swinging of the PAC by vigorous cardiac contractions can lead to a recording artifact in which the PA systolic pressure is falsely elevated and the PA diastolic pressure is falsely decreased.

III. ARTERIAL PRESSURE MONITORING

Arterial blood pressure is probably the most frequently used parameter to guide therapy in the critically ill patient. Several techniques, both invasive and non-invasive have been developed to measure and monitor blood pressure in the
intensive care unit. The invasive method refers to an intra-arterial catheter that allows direct and continuous measurement of the blood pressure. Non-invasive methods are divided into manual and automated methods, the latter of which is more commonly used in modern ICU’s.

Non-invasive Blood Pressure Monitoring

The basic principle behind BP measurement was introduced in Italy in 1896 by Riva-Rocci. This principle involved applying a pressure over the artery until it is completely compressed and then relieving the pressure slowly until the artery starts to open. The recording of the first opening of the artery reflects the systolic blood pressure.

There are several manual methods of detecting the opening of the artery, most commonly is by auscultation of Korotkoff sounds. Korotkoff sounds are low frequency sounds (20-50 Hz) that reflect vibrations of the arterial wall upon resumption of flow. The appearance of Korotkoff sounds heralds systolic BP (SBP) and the disappearance, or more accurately, the muffling of the sounds indicates the diastolic BP (DBP). Because of their low frequency, Kortkoff sounds can be difficult to hear especially in loud surroundings.

Other ways include the oscillation method, in which a pulsatile movement of the mercury column or the needle of the aneroid manometer represents the systolic blood pressure. Sometimes, feeling for the resumption of blood flow through the distal branches of the compressed artery is used to assess the systolic pressure especially in emergency situations (palpation method).

Automated methods refers to the inflation and deflation of the cuff by machine rather than manually. Recording of the blood pressure is also done by the machine and there are different kinds of machines based on the signal they record. Doppler flow machines record the changes in the reflected echo signal in the distal artery during inflation and deflation of the cuff. The appearance of the Doppler shift indicates systolic pressure and disappearance of the Doppler shift indicates diastolic pressure. The infrasound system uses a microphone to record the sounds that results from arterial wall vibrations due to blood flow (similar to Korotkoff sounds). Similarly, oscillations of the arterial wall can also be recorded by a special sensor that uses the principle of plethysmography to detect blood pressure changes in the underlying artery. This is called the oscillometric method. Another method is based on the pulsatile unloading of the finger arterial walls using an inflatable finger cuff with a built-in photoelectric plethysmograph. While continuously measuring the blood pressure, this monitor calculates cardiac output as well.

Invasive Blood Pressure Monitoring

Indications:

Invasive monitoring of the blood pressure was developed in order to overcome the obvious shortcomings of non-invasive BP measurement. The invasive technique allows for direct rather than indirect measurement of the blood pressure in a continuous fashion. This exact and continuous measurement becomes invaluable in cases of blood pressure extremes especially when considering intravenous continuous medication infusion such as vasodilators or vasopressors. In addition to providing blood pressure information, the arterial line provides an easy access to draw arterial blood gas and other blood tests to monitor acid-base disorders and oxygenation in the ventilated patient.

Contraindications:

As with all vascular accesses, arterial lines have site specific and general contraindications. Site-specific contraindications include local infection, full thickness burns that have an increased risk of infection and the presence of a vascular prosthesis or a hemodialysis fistula in the same extremity. General contraindications include uncorrected bleeding diathesis or a recent use of thrombolytics. Those contraindications are relative and generally avoidable.

A specific contraindication to arterial lines is the presence of limb ischemia. The famous Allen and modified Allen tests are generally recommended before attempting radial arterial puncture to avoid the devastating event of forearm ischemia due to damage to the only functioning arterial branch.

Allen and Modified Allen tests:

In the modified Allen test, the examiner holds the patient’s hand up to prevent venous blood pooling towards the forearm. The patient is then asked to form a fist and the examiner compresses both the ulnar and radial arteries at the same time. The patient then lowers his hand and opens his fist. A pale palm is noted; at this point the examiner releases the ulnar artery and observes for red coloration of the palm within 6 seconds. If this coloration takes longer than 10 seconds the test is considered abnormal. This test assesses the sufficiency of the ulnar circulation in case of damage to the radial artery during the catheterization.

The original Allen test involves the same maneuvers but is performed to assess the sufficiency of both the radial and ulnar arteries by compressing the other artery each time.

The Allen test can be performed using the pulse oximeter waveform deformation technique as well. In this technique the pulse oximetry sensor is placed on a finger in the examined
hand. The examiner occludes the ulnar and radial arteries until the pulse oximetry waveform disappears. Subsequently, each artery is released at a time and the waveform is observed. The reappearance of a normal or slightly blunted waveform indicates the adequacy of flow through the released artery.

**Insertion Technique:**

Regardless of which artery will be used to establish an arterial line, the same principles generally apply. First, the skin overlying the puncture site needs to be aseptically cleansed, then the operator localizes the pulsating artery with the index finger of the non-dominant hand. The direction of insertion is determined by feeling for the artery with the third finger and the point of entry is just underneath the index finger. Only gentle pressure should be applied as the artery might collapse easily especially if the arterial blood pressure is low. Arterial punctures are generally more painful than venous ones, and occasionally applying a local anesthetic to the area surrounding the puncture site allows for easier access as it prevents arterial spasm especially if the radial approach is used.

Either of two techniques can be used to obtain the arterial access depending on the location of the guide-wire. In the first, the integral guide-wire technique, the needle carries the catheter on its outer surface and within it there is a built-in guide-wire that can be advanced to secure the arterial access once a blood flush is seen. The catheter is then advanced over the needle and the guide-wire, both of which are removed once the catheter is in place. Most commercial radial “A-line” kits use the integral guide-wire technique. In the second technique, a needle-catheter combination is used to enter the artery. Once the artery is punctured, the needle-catheter combination is advanced slightly before the needle is withdrawn, leaving only the catheter in place. A pulsating blood flow should be noted before advancing a separate guide-wire through the catheter. Once the guide-wire is in place, the catheter is fully inserted and secured. If a pulsating blood flow is not seen after withdrawing the needle, the catheter is withdrawn slowly until a pulsating flow is seen and the guide-wire is then advanced as mentioned above.

The intra-arterial catheter needs to be secured carefully, usually with sutures and then connected to a leveled pressure transducer to allow pressure monitoring.

There are four placement locations for the arterial line. The radial artery is by far the most commonly used followed by the femoral artery. The *dorsalis pedis* is used less commonly and the brachial artery is generally avoided unless other sites do not work due to its proximity to the median nerve and paucity of collaterals. It is important to remember that with femoral artery line placement, the patient needs to remain supine. Moreover, *dorsalis pedis* artery wires generally record systolic blood pressures that are 5-20 mmHg higher than radial arterial measurements. Using the mean arterial pressure (MAP) rather than systolic or diastolic blood pressures carries the advantage of avoiding blood pressure variations secondary to variations in the distance from the aorta. The further the artery from the aorta, the higher the systolic pressure due to the more pronounced pressure waves reflected back from the periphery. In spite of this, the mean arterial pressure does not change with distance from the aorta because the increased peak systolic pressure is offset by the narrowing of the pressure wave. This aspect and the fact that the MAP is the actual driving force of the blood to supply the organs, make the MAP the preferred measurement in the ICU.

**Invasive vs. Non-invasive Blood Pressure Monitoring**

Invasive blood pressure monitoring has limitations that justify the search for the optimal noninvasive blood pressure monitoring technique. These include risk of infection and bleeding, as well as the pain and cost associated with the technique. In addition, the invasive method has its own flaws specifically related to recording artifacts in the form of waveform distortion. In order for the recorded arterial pressure to be valid, a normal waveform needs to be obtained. An underdamped waveform is suspected when the systolic BP peak is sharp and this is usually due to long tubing system between the catheter and the pressure transducer. In contrast, an attenuated waveform with slow upslope and downslope fluctuations is secondary to overdamping of the system, which is usually due to air bubbles trapped in the tubes or within the pressure transducer. An underdamped system can overestimate the SBP and the overdamped system can underestimate it. The flush test is occasionally used to differentiate a normal tracing from underdamped or overdamped one. Certainly, there is doubt regarding the reliability of non-invasive methods in the critical care setting, justifiably so. It has been shown that in cases of low flow states and hypotension, non-invasive measurement significantly underestimates blood pressure. Some authors have attributed failure of non-invasive techniques to accurately correlate with invasive pressures to “miscuffing” i.e. choosing the wrong cuff size. Nevertheless, recent studies have repeatedly demonstrated this lack of reliability despite optimal “cuffing”. Even small deviations from true blood pressure can be deleterious in the intensive care settings as hemodynamic interventions can be started or stopped based on a single number like the mean arterial pressure (MAP) one example is the goal-directed therapy for septic shock.

Therefore, it seems reasonable at this time to use invasive blood pressure monitoring in all unstable or potentially unstable patients regardless of etiology.
IV. CENTRAL VENOUS CATHETERS

The use of central venous catheters (CVCs) in the modern ICU has increased dramatically over the past few decades. The Center for Disease Control and Prevention (CDC) estimates that each year there are 15 million days of CVCs use in the ICUs of the U.S alone. This high volume is justified by the wide array of diagnostic and therapeutic potentials of the CVCs.

By definition, a catheter is considered to be central venous if it is placed in either of the internal jugular, subclavian or femoral veins. Those catheters are generally large and may have a single or multiple lumens. Heparin-bonded catheters and antibiotics–impregnated catheters are available.

Indications

As mentioned above, the CVCs have a significant number of functions, most of which are listed below:

1- Monitoring the central venous pressure (CVP).
2- Rapid administration of resuscitative fluids.
3- Administration of caustic materials such as chemotherapeutic agents or irritating electrolytes (e.g. potassium chloride at high rates).
4- Administration of vasoactive medications (vasopressors) that can lead to local complications if administered in a peripheral vein in case of extravasation.
5- Administration of hypertonic fluids and total parenteral nutrition (TPN).
6- Long term venous access such as for the administration of long courses of antibiotics or home inotropic support.
7- Access for placement of pulmonary artery catheters and temporary transvenous pacemakers.
8- Access for hemodialysis or plasmapheresis.

Contraindications

The only absolute contraindication for the placement of CVCs is refusal by a competent patient or guardian. Other contraindications are relative and are divided into site specific and general vascular access contraindications.

The site-specific contraindications include:

- Chest wall deformities that preclude the insertion of a subclavian CVC.
- Poor pulmonary reserve that renders an iatrogenic pneumothorax (in the subclavian and internal jugular CVCs) a considerable risk.
- Superior vena cava clot or mass that prevents a successful insertion (subclavian and internal jugular CVCs).
- Presence of a pacemaker or defibrillator lead in the same vein should be avoided if possible.

General contraindications include an unfavorable anatomy (from previous trauma or surgery) and a patient who is unable to stay still during the procedure, which will present a significant risk of complications in case of an unexpected movement during the procedure. Light, conscious sedation and adequate pain control usually allow the operator to proceed.

The presence of coagulopathy have been studied and is felt not to be a contraindication to the placement of central venous catheters. Nonetheless, in non-urgent cases, correction of a significant bleeding diathesis is considered a safe practice.

Insertion Technique

After reviewing the chart and obtaining informed consent, a time-out is taken in which the patient’s name, date of birth or medical record number, the type of the procedure and the location of the procedure, are reviewed and communicated verbally with loud voice by the staff involved in performing and monitoring the procedure. An experienced operator should be present throughout the procedure. The operator should wash his hands prior to and after the procedure. Full barrier precautions that include face mask, gowns, and sterile drapes are mandatory. Only sterile gloves may be used.

Subclavian vein access

The subclavian vein originates from the axillary vein and ends posterior to the medial head of the clavicle where it joins the internal jugular vein to form the brachiocephalic vein. The
right brachiocephalic is shorter and more straight that the left one. With the patient in the reverse Trendelenburg position, the operator identifies the medial and middle thirds of the clavicle and inserts the needle 1 cm underneath their junction. The needle should be directed towards the suprasternal notch. This is the inferior route and is most commonly used.

**Internal jugular vein access**

The internal jugular vein runs in the neck within the carotid sheath, which also includes the carotid artery and the vagus nerve. The internal jugular vein is commonly approached through one of two locations that are marked by the sternocleidomastoid (SCM) muscle. The **middle approach** uses the triangle formed by the two bellies of the SCM and the clavicle as a landmark. The needle is inserted at the apex of the triangle and is directed towards the ipsilateral nipple. If continuous negative pressure is maintained while inserting the needle, a blood flush indicating intravascular location of the needle, is obtained at a depth of 2-4 cm. Caution must be exercised when palpating the carotid artery for positioning because significant compression can collapse the vein and make accessing it difficult, with the resultant risk of mistakenly accessing the artery. The posterior approach is sometimes used; this starts by identifying the two bellies of the SCM muscle, the lateral belly with the thumb of non-dominant hand, and the medial belly by the index finger of same hand. The needle entry point is just above the point where the external jugular vein traverses the lateral belly of the SCM. The needle is inserted underneath the lateral belly and directed towards the suprasternal notch.

The use of real-time ultrasound guidance during the insertion can reduce the rate of complications and improve the rate of successful first attempts of the internal jugular and subclavian vein access.  

**Femoral vein access**

In the groin, the femoral vein lies medial to the femoral artery and nerve. The femoral artery pulse is used as a landmark and the needle is inserted medial and slightly inferior to it at a 45 degrees angle, 2-3 cm below the inguinal ligament.

**Which access to choose?**

Each of those venous accesses carries site specific risks that make them more or less favorable depending on the clinical situation. Overall, the CDC reports that the subclavian approach is the best location given the lower risk of infections as well as is more convenient in an awake patient as opposed to other approaches. Ultimately, the approach with which the operator feels more comfortable is the one he or she should choose. Table 3 delineates the relative risks of each approach.

**Clinical significance**

CVCs allow for the determination of central venous pressure (CVP) and this pressure has been traditionally used as a surrogate of the right ventricular preload and overall intravascular volume. It has been stipulated that the absence of respiratory variations in the CVP waveform predicts a lack of response of the cardiac output to further fluid administration. To minimize the effect of intrathoracic pressure variations with respiration, the CVP should always be measured at the end of expiration when the extravascular pressure (intrathoracic pressure) is closest to zero.

The CVCs have been compared favorably to the pulmonary artery catheter for the management of acute respiratory distress syndrome (ARDS) patients. Moreover, the CVP catheter has been shown to be useful in the initial goal-directed therapy protocol for patients with septic shock. In the first 6 hours of presentation to the ED, the protocol involves maximizing the CVP to > 8 mmHg and then using vasoactive medications to achieve a MAP > 65 mmHg. Once the MAP is stable > 65 mmHg, then the goal becomes to maintain central venous oxygen saturation > 70 % by transfusing red blood cells if the hematocrit is < 30% and by using inotropes if the hematocrit was > 30 %. The early goal directed therapy has demonstrated its superiority to the regular management of septic shock patients.

**REFERENCES**


