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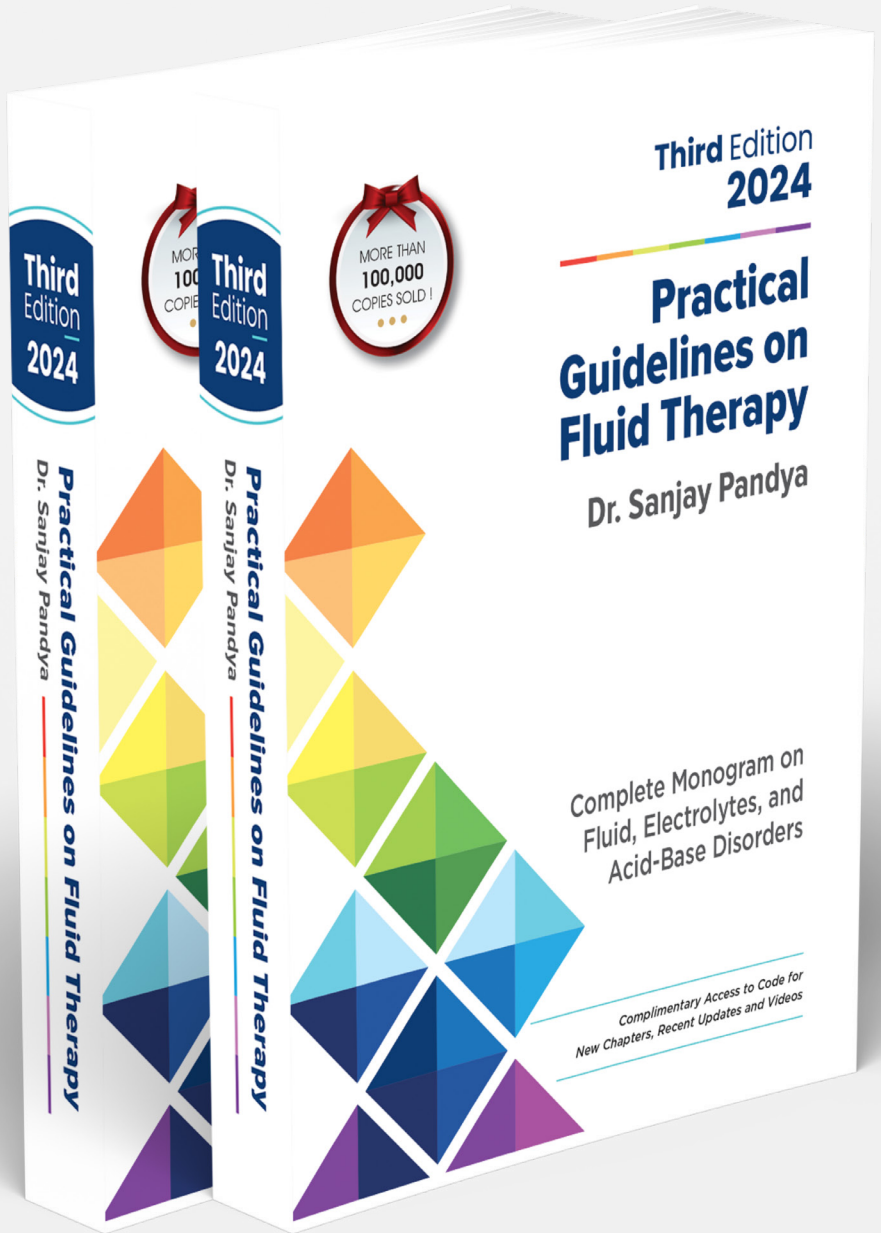
Chapter 17:

Static Hemodynamic Monitoring Techniques



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17 Static Hemodynamic Monitoring Techniques

Commonly used static hemodynamic monitoring methods are inferior vena cava assessment, central venous pressure (CVP) monitoring, arterial cannulation, and pulmonary artery catheter monitoring. Although these modalities are available more widely than compared to dynamic monitoring modalities, they lack precision and accuracy.

INFERIOR VENA CAVA ASSESSMENT

Echocardiography of the inferior vena cava (IVC) is a simple, routinely used, noninvasive tool that can rapidly measure maximal IVC diameter and respiratory variations in IVC diameter at the bedside for predicting the intravascular volume status and fluid responsiveness [1]. For the initial evaluation of shock, as compared to more invasive technologies, this modality is preferred to assess the hemodynamic status and fluid responsiveness [2, 3].

For the proper understanding clinical utility of this tool, it is discussed step by step as follows:

- Physiological variations in IVC diameter during the respiratory cycle.
- Measurement techniques.

- Maximal IVC diameter in spontaneous breathing and patients on a ventilator.
- Inferior vena cava diameter variation (IVC distensibility index (dIVC) in patients on mechanical ventilator and IVC collapsibility index (cIVC) during spontaneous breathing).

The diagnostic value of the variability of the superior vena cava is better than that of the IVC [4], but the limiting factor is a requirement of transoesophageal echocardiography for the measurement, which is minimally invasive and technically difficult [5].

A. Physiological principles

It is important to remember that amongst patients with spontaneous respiration, and in patients on positive pressure ventilation, physiological changes, values of variations of IVC parameters, and their interpretations differ markedly.

1. IVC in spontaneous respiration

In patients with spontaneous breathing, intrathoracic pressure decreases during inspiration, which increases the venous return of blood, resulting in the "collapse" of the vena cava. Inversely, intrathoracic pressure increases during expiration, which reduces the venous return of blood and thus causes the expansion of the vena cava.

IVC collapsibility index (cIVC): "Collapsibility index" is used to assess the respiratory variations in IVC diameter in patients with spontaneous respiration.

Calculation of the IVC collapsibility index: In spontaneous breathing patients, obtain the maximum IVC diameter (at the end of expiration) and minimum IVC diameter (at the end of inspiration) and calculate the index.

$$\text{IVC Collapsibility Index} = \frac{\text{Maximum IVC} - \text{Minimum IVC}}{\text{Maximum IVC}} \times 100$$

2. IVC in patients on ventilator

In patients on positive pressure ventilation (on noninvasive ventilation or intubated and on the mechanical ventilator), IVC changes with respiration are just reversed compared with spontaneous breathing. During inspiration, intrathoracic pressure increases, which pushes blood back into the IVC from the right atrium causing "Distention" of IVC. While during expiration, intrathoracic pressure decreases, and venous return increases, which causes IVC collapse.

IVC distensibility index (dIVC): "Distensibility index" is used to assess the respiratory variations in IVC diameter in patients on a ventilator.

Calculation of the IVC distensibility index and IVC diameter variability: In patients on a ventilator, obtain the maximum IVC diameter (at the end of inspiration) and minimum IVC diameter (at the end of expiration) and calculate the mean of both (mean IVC), and calculate indexes [6].

$$\text{IVC Distensibility Index} = \frac{\text{Maximum IVC} - \text{Minimum IVC}}{\text{Minimum IVC}} \times 100$$

$$\text{IVC Diameter Variability} = \frac{\text{Maximum IVC} - \text{Minimum IVC}}{\text{Mean IVC}} \times 100$$

Why is the predictive value of variations in IVC diameter better in patients on a ventilator than in spontaneous breathing patients?

In spontaneous breathing patients, respiration is highly variable between different patients, and it varies from one to another respiratory cycle, even in the same patient. Additionally, in spontaneous breathing patients, changes in intrathoracic pressure are much smaller, and therefore variation in the size of the IVC is also smaller. So, during spontaneous breathing, respiratory variations in IVC diameter are nonuniform and

smaller; therefore, its predictability for fluid responsiveness is inferior [7].

While in a patient on a ventilator, controlled large tidal volume and small positive end-expiratory pressure lead to uniform changes in intrathoracic pressure during the respiration cycle. As a result, changes in vena cava diameter are also uniform (without variability from one to another cycle of ventilation), and therefore, patients on ventilators predict fluid responsiveness better than spontaneous breathing patients.

B. Measurement techniques

Two types of techniques used for the IVC measurement are:

- Transthoracic echocardiography (TTE): The diameter of the inferior vena cava can be quite easily and routinely measured by using transthoracic echocardiography using a subcostal view in a longitudinal section (measured approximately 1–2 cm caudal to the right atrium and IVC junction).
- Transesophageal echocardiography (TEE): TEE provides an accurate view of the superior vena cava (SVC) and, therefore, provides more precise data for the calculation but is used infrequently and in selected patients as this method is minimally invasive (requires intubation) with many limitations.

C. Maximal IVC diameter

Assessment of maximal IVC diameter is a static parameter that may be used to evaluate volume status and fluid responsiveness in spontaneously breathing patients [2]. In patients with shock, despite normalization of blood pressure after fluid resuscitation, inadequate dilatation of the IVC may be an important clue for insufficient circulating blood volume [8]. However, clinicians should use static IVC diameter cautiously because of its poor predictability [9]. Maximal IVC diameter is not useful in most patients, but very low or very high values may be helpful [10].

1. In spontaneous breathing

- Complete IVC collapse or end-expiratory IVC diameter less than 10 mm may suggest hypovolaemia and needs fluid administration in spontaneously breathing patients [11–14].
- IVC diameter of more than 25 mm may suggest hypervolemia in spontaneously breathing patients, and fluid administration is not appropriate [11, 15].
- In patients with marked volume overload, IVC is expanded and not collapsible [16]. However, noncollapsing large IVC diameter is not specific for volume overload, and it is essential to exclude multiple other causes [15].

2. In mechanically ventilated patients

A single static value of IVC diameter does not predict fluid status or fluid responsiveness in patients on a ventilator [6]. It is important to remember that IVC is often distended in patients on a mechanical ventilator due to increased intrathoracic pressure because of positive pressure ventilation. Therefore, a distended IVC in patients on a ventilator neither suggests volume overload nor excludes hypovolemia or fluid responsiveness.

However, an end-expiratory IVC diameter of less than 13 mm is a strong indicator of volume depletion, and an IVC diameter over 25 mm excludes fluid responsiveness in patients on a mechanical ventilator [10, 17].

Pitfalls: Between the vena cava and right atrium, there is no valve, and

therefore, high right atrial pressure causes expansion of the vena cava and impairs the normal collapsibility of the vena cava. So, before diagnosing the volume status based on IVC parameters, consider other factors that can affect the IVC diameter and collapse, such as right atrial pressure, respiratory disorders affecting the degree of intrathoracic pressure change, and pressure within the abdominal cavity.

Remember that prerequisites to use IVC diameter and collapsibility to assess volume status effectively are controlled mode ventilator, tidal volume ≥8mL/kg with positive end-expiratory pressure (PEEP) ≤5 cm H_2O, intraabdominal pressure should be normal, and absence of acute cor pulmonale or severe right ventricular dysfunction [14, 15]. Fluid responsiveness is predicted poorly if tidal volumes <8 mL/kg or PEEP >5 cm H_2O [18].

D. Inferior vena cava diameter variation

In spontaneously breathing patients, the average diameter of IVC is about 18 mm, with approximately 50% collapse with inspiration [19].

Assessment of the change in diameter of the vena cava during respiration and calculation of the distensibility/collapsibility index on its basis is a widely used tool for predicting intravascular volume status and fluid responsiveness. Variations in IVC diameter in response to fluid administration is a better indicator of adequate replacement of fluid than vital signs, and static maximal IVC diameter [9].

For the assessment of fluid responsiveness, the predictive value of variations in IVC diameter is better in patients on a ventilator compared to patients on spontaneous breathing [20, 21].

The measurement of inferior vena cava diameter is routinely performed in ICUs for both patients spontaneously breathing and on a ventilator. But it is important to remember that evidence about the predictive value of this modality in the assessment of volume status and fluid responsiveness in both groups is conflicting and the subject of debate [3, 17, 22–24].

1. In mechanically ventilated patients

There are controversies regarding the effectiveness of respiratory variation in IVC diameter and distensibility index to predict fluid responsiveness in mechanically ventilated patients. There are studies, systematic reviews, and meta-analyses supporting its predictive value and use [18, 20, 21, 25–32], and data not in favor of the same [4, 10, 33–35].

The distensibility index (dIVC) is a more accurate parameter in predicting the volume status as compared to the collapsibility index (CI-IVC) and variations in IVC (ΔIVC) in patients with positive pressure supports [36].

In patients on a ventilator:

- If the distensibility index is >18% or IVC diameter variability is >12%, it may predict fluid responsiveness [25–27].
- If the distensibility index is <18%, the patient is not responsive to volume, and benefit from fluid administration is unlikely [37].

In patients on a ventilator, as compared to the measurement of IVC collapsibility, SVC collapsibility has better diagnostic accuracy in predicting fluid responsiveness [5]. The SVC collapsibility threshold of 36% can effectively differentiate between fluid responders and nonresponders patients (with a sensitivity of 90% and a specificity of 100%)

[38]. But the measurement of SVC needs semi-invasive transesophageal echocardiography, which is the limiting factor.

2. In spontaneous breathing patients

Because of conflicting evidence, use IVC diameter variation and collapsibility index cautiously to predict fluid responsiveness in spontaneously breathing patients. Evidence supports its predictive value and uses [7, 9, 20, 39–44], and there is also literature not favoring the same [21, 45–48]. Remember that optimal thresholds of these parameters in spontaneous breathing are different from those described in patients on a ventilator [39].

In spontaneous breathing patients:

- Very collapsible IVC with higher amplitude respiratory variation, i.e., collapsibility index >40%, is a moderate predictor of fluid responsiveness [7, 40–42], but lower values (<40%) do not exclude volume responsiveness [9, 19].
- Very distended IVC with a collapsibility index <15% may suggest that the patient is not fluid responsive [49].
- The absence of respiratory variation is an important clue, which suggests that the patient is not fluid responsive [26].

Besides low sensitivity and less reliability in patients with spontaneous breathing, cautious use of this modality is preferred because it is readily available, easy to perform, noninvasive, and carries high specificity [40, 43, 44].

CENTRAL VENOUS PRESSURE MONITORING

Central venous pressure is the most frequently used method to assess the hemodynamic status and guide fluid resuscitation in ICU patients [50, 51].

What is CVP?

Central venous pressure is the pressure of blood measured in the vena cava at its junction with the right atrium. CVP is a simple method that can be easily measured in any patient with a central venous line, and the most frequently chosen access is the internal jugular vein.

CVP measures mean right atrial pressure and is an indicator of chiefly right ventricular preload and, to a lesser extent, left ventricular preload.

Factors determining CVP

CVP is influenced by multiple factors such as vascular volume status, right ventricular compliance, pulmonary vascular resistance, thoracic, pericardial, and abdominal pressures, peripheral vascular tone, and posture [11, 52]. In patients on a mechanical ventilator, the value of CVP increases proportionate to positive end-expiratory pressure (roughly 5 cmH_2O increase in PEEP will cause a 2.5 cmH_2O increase in CVP) [53].

CVP measurement is unreliable in the presence of pulmonary vascular disease and hypertension, right ventricular disease, congestive heart failure, valvular heart disease, tense ascites, and high intra-abdominal pressure.

Interpretation of the value of CVP

The value of CVP should always be interpreted with clinical status.

Normal value

The normal value of CVP is 2 to 6 mmHg (when measured continuously using electronic pressure transducers) or 3 to 10 cm H_2O (measured directly using water manometers).

Low CVP

- True hypovolemia, as in blood loss, fluid loss, or fluid shift.
- Relative hypovolemia caused by peripheral vasodilatation, as in spinal anesthesia, septicemia, and anaphylactic shock.

High CVP

- Volume overload.
- Cardiac causes like congestive heart failure, cardiac tamponade, constrictive pericarditis, and tricuspid regurgitation.
- Pulmonary causes like embolism, pulmonary hypertension, tension pneumothorax, COPD, and cor pulmonale and positive pressure ventilation.

When and how to use CVP in clinical practice?

In the past, CVP monitoring was the most commonly used tool to assess volume status and to guide fluid resuscitation in critically ill patients [54]. However, in recent literature, the relationship between CVP and blood volume was poor, and CVP was unable to predict the hemodynamic response to a fluid challenge [2, 55, 56].

With recent evidence, the concept of using static marker CVP to assess intravascular volume or an indicator of fluid responsiveness is not recommended and is considered unreliable and potentially dangerous [57, 58].

However, CVP is still used in ICUs worldwide because other more accurate methods are not available easily [54].

In view of two contradictory facts (i.e., growing literature against the use and routine worldwide use in practice), different practical aspects related to indications and limitations of CVP monitoring are summarized.

Basic principles

- Normal CVP does not exclude volume depletion.
- A single value of CVP does not help in managing patients; it is always interpreted in the context of the clinical situation [57, 59].
- The "extreme" CVP values are important; it provides valuable guidance. The presence of "extreme" CVP values (CVP <6–8 mmHg and CVP >12–15 mmHg) can be of great help in predicting volume status and planning fluid administration [60–62].
- Using CVP to guide fluid administration is far from perfect, but when more accurate predictors of fluid responsiveness are not available, a reasonable CVP target is 8–12 mmHg [62, 63] during fluid administration. However, if the patient is stable, no attempt should be made to increase CVP to specific target values. Instead, the upper limit of CVP should be determined individually, considering the potential benefit/risk of further fluid administration. The goal is to keep CVP as low as possible while maintaining adequate tissue perfusion [62].

Low CVP

- A low CVP can be normal but generally suggest hypovolemia. If CVP is normal, it does not exclude hypovolaemia [64].
- In patients with low CVP values (less than 6 mmHg), an initial moderate fluid bolus is unlikely to cause harm, and most patients will respond to fluids [59–62].
- Prognostic value: In patients with circulatory shock, lower CVP and increased cardiac output may improve the prognosis and renal function, so one should try to keep lower CVP with

due caution to maintain adequate tissue perfusion [62, 65].

High CVP

- High CVP is always abnormal and has important therapeutic and prognostic values.
- An increase in CVP can occur due to increased total vascular volume, decreased cardiac function, or both.
- Patients with high CVP values (greater than 15 mmHg) do not respond to fluid administration, and therefore it is prudent to avoid the administration of fluids when the CVP is markedly elevated [61, 62].
- The upper threshold of CVP can alert the clinician to stop fluid therapy, and a positive trend reaching high values (CVP >8 mmHg) may warn the clinician that fluid replacement is no more needed [64]. So, the use of high CVP as a safety end-point and as a stopping rule for fluid administration can minimize the risk of volume overload [66].
- Prognostic value: Elevated CVP (>10 mmHg) is associated with an increased risk of mortality, a higher incidence of acute kidney injury (AKI), and poor outcomes (i.e., death) in critical ICU patients [65, 67–71]. A high CVP causes an increase in renal venous pressure leading to increased renal venous congestion, and reduces renal perfusion pressure with AKI as a result [72]. Similarly, elevated venous pressure may impair the venous return and disturb microcirculatory blood flow, which may harm the functions of different organs, leading to poor outcomes and even high mortality [58, 68].

Conclusion: Except for the extreme values, CVP may provide inaccurate estimations of volume status and does not predict volume responsiveness. When more accurate methods to estimate a patient's fluid volume status are unavailable, clinicians should understand the limitations, dangers, and benefits of CVP and use it judicially and selectively rather than abandon it altogether.

ARTERIAL CANNULATION

Arterial cannulation is a frequently performed and preferred procedure for the hemodynamic monitoring of critically ill and high-risk surgical patients [64].

Use

Arterial catheterization is useful for:

1. **Continuous monitoring of arterial blood pressure:** Arterial catheterization is a low-risk method which provides reliable information about blood pressure in unstable patients. This beside technique continuously and accurately measures beat-to-beat (as well as moment-to-moment) blood pressure and therefore recognizes the changes in blood pressure promptly and guides clinicians for quicker therapeutic interventions.
2. **Frequent blood sampling:** Arterial catheterization helps in repeated blood sampling for laboratory testing or arterial blood gas analysis.
3. **To predict fluid responsiveness:** An arterial catheter allows the analysis of arterial pressure waveforms. Analysis of respiration variations in the arterial waveform can determine fluid status and fluid responsiveness by calculating indices such as pulse pressure variation (PPV) and stroke volume variation (SVV). In addition, variations in the arterial waveform in response to fluid challenges or

passive leg raising are reliable indices for assessing fluid responsiveness.

4. **Diagnostic or therapeutic interventions:** Arterial catheterization is used for various diagnostic and therapeutic coronary procedures such as vascular stenting or embolization and intra-aortic balloon pump (IABP).

Technique

Arterial cannulation is a relatively safe procedure in which a simple cannula is introduced by a Seldinger technique, usually in a radial or femoral artery, after local anesthesia. Hemorrhage or hematoma at the puncture site, arterial spasm or occlusion, embolization, distal ischemia, and local and catheter-related infection are common complications of arterial cannulation. Avoid using arterial lines to administer medication, as it can lead to serious tissue damage and cause considerable morbidity [73].

Indications

Common indications of arterial cannulation for hemodynamic monitoring are:

- In ICU for monitoring of critically ill patients with circulatory shock.
- Major surgery such as cardiothoracic surgery or major abdominal surgery.
- When noninvasive blood pressure measurements are unreliable or difficult such as with severe burns or trauma.

Contraindications

Common contraindications of arterial cannulation are:

- Severe peripheral vascular diseases, arterial atherosclerosis, insufficient collateral perfusion, or absent pulse.
- Anticoagulation therapy or the presence of coagulopathy.

Radial or femoral, which artery to select for the cannulation?

The radial artery is a common choice because of easy cannulation due to its superficial position, the adequate collateral blood supply to the hand via the ulnar artery (reduces the risk of hand ischemia due to catheter induced thrombosis of radial artery), low rates of complications, easy to compress for the control of bleeding during cannulation or following its removal, and early ambulation [74–77]. Perform the modified Allen test before cannulation of the radial artery to assess the collateral circulation to the hands.

The limitations of the radial arterial cannulation approach are:

- Significantly smaller average lumen diameter (less than 3 mm) makes the trans-radial approach difficult in small-sized patients.
- Higher risk of thrombosis and nerve injury.
- Locating the radial artery via palpation may be difficult in patients with severe hypotension, morbid obesity, and weak pulse due to atherosclerosis.
- In patients with severe peripheral vasoconstriction and sicker patients on higher vasopressor doses, the value of radial arterial blood pressure is lower and may underestimate the central blood pressure [78–80].

The femoral artery is the second most common cannulation site [81], but the trend to use the femoral artery first is increasing in many patients with critical illness [80] because:

- It is easiest to cannulate and therefore provides faster access during emergencies in patients with shock. In severely hypotensive patients with nonpalpable peripheral pulses,

greater ease in locating and cannulating due to its large lumen.

- There may be discrepancies between radial and femoral arterial blood pressure measurements. However, patients with severe hypotension on higher vasopressor doses and hypothermia femoral approach provide accurate blood pressure measurement [82, 83]. So, the femoral approach of arterial cannulation is beneficial in high-risk patients undergoing longer surgical interventions and helps to avoid inappropriate administration of vasopressors and/or inotropic agents [84].
- More reliable functioning and less likely to fail because of inaccuracy, blockage, or accidental removal [85].
- The radial artery is thin, not palpable, or there may be contra-indication to intra-arterial catheter placement at the wrist in certain patients [86].

The limitations of the femoral arterial cannulation approach are:

- Higher risk of arterial catheter-related infection [87, 88].
- Limits mobility significantly, and in alert patients, delays ambulation.
- Difficult to control or prevent bleeding (unlike radial artery), carries a great risk of retroperitoneal hematoma, and its diagnosis is often delayed [86].

PULMONARY ARTERY CATHETER MONITORING

The pulmonary artery catheter (PAC) is an invasive diagnostic procedure in which a catheter is inserted through a central vein into a pulmonary artery. PAC was used widely and considered the gold standard for measuring cardiac output in the past. But currently, PAC is used sparingly for the management of critically ill or perioperative patients [89].

Description

A pulmonary artery or Swan-Ganz catheter is a pliable catheter made from polyvinyl chloride material with a four lumen and a thermodilution sensor.

Two ports, including the Red port used for balloon inflation and the Yellow port, along with the thermodilution sensor, open within the pulmonary artery lumen, while the remaining two ports, the Proximal (Blue) port, and the White port (Clear lumen), open within the right atrium, as summarized in Table 17.1.

Technique

For the measurement of pulmonary artery occlusion pressure (PAOP), the pulmonary artery or Swan-Ganz flexible balloon-tipped, flow-directed catheter is introduced through a large vein and is placed with the tip in a distal pulmonary artery. PAC is an invasive multi-lumen central line placed through a large vein such as an internal jugular, subclavian, or femoral vein.

By using a flow-directed balloon flotation technique and utilizing pressure waveform and or fluoroscopy/echocardiographic guidance, PAC traverses along with venous blood into the vena cava (superior or inferior), right atrium, tricuspid valve, right ventricle, pulmonic valve and finally placed in the pulmonary artery [90]. Continuous monitoring of catheter tip pressure provides clues about the position of the tip of the catheter that guides the clinician to traverse the catheter to the pulmonary artery. In addition, the X-ray chest and wedge pressure waveform helps to confirm the location of PAC [91].

What information does PAC provide?

PAC provides important information by directly measuring cardiac pressures

Table 17.1 Sensor and lumens of pulmonary artery catheter			
	Location	**Description**	**Function**
Thermodilution sensor	Within the pulmonary artery lumen	Located 4 cm from the tip, proximal to the balloon	It measures the blood temperature in the pulmonary artery and assists in calculating cardiac output using the thermodilution technique
Lumen or ports			
Red port Balloon inflation port	Within the pulmonary artery lumen	It terminates in the balloon at the tip of the catheter, located approximately 2 cm from the distal end	The red port is used solely for inflating and deflating the balloon by using a custom syringe and air
Thermistor lumen Distal port Yellow port		The distal port terminates at the tip of the 110 cm long catheter, which should be positioned in the pulmonary artery	The yellow port useful for measuring pulmonary artery pressure, mixed venous samples, and allows continuous cardiac output monitoring
Proximal lumen Blue port CVP port	Within the right atrium	Located 30 cm from the tip of the catheter and rests within the right atrium	It is utilized for measuring and monitoring CVP, right atrial pressure as well as for fluid and drug administration
White port Clear lumen		Located 31 cm from the tip of the catheter and rests within the right atrium	The white port is used for infusing fluids and drugs

(central venous pressure, right atrial and ventricular pressures, pulmonary arterial pressure, pulmonary artery occlusion pressure) and cardiac output [57, 92]. In addition, PAC also indirectly measures systemic and pulmonary vascular resistance, cardiac index, stroke volume, oxygen delivery, and mixed venous oxygen saturation (SvO_2).

1. Pulmonary artery occlusion pressure (PAOP)

Also known as pulmonary arterial wedge pressure (PAWP) or pulmonary capillary wedge pressure (PCWP), is a technique to measure pulmonary venous pressure and left atrial pressure indirectly by using PAC. In this method, the tip of a PAC is placed into a smaller, more distal pulmonary arterial branch. When the balloon at the distal tip of the PAC is inflated, it obstructs the forward blood flow in the distal branch of the pulmonary artery, and subsequently, measurements are obtained.

The pulmonary artery is in direct continuity with the left atrium and, therefore, with the left ventricle during diastole. So, PAOP, in addition to measurement of left atrial pressure, during diastole, reflects left ventricular end-diastolic pressure (LVEDP) and determines left ventricle (LV) function when mitral valve function is normal.

2. Cardiac output

PAC measures CO invasively using the thermodilution principle and was considered the gold standard method. Details about the same are covered in the invasive systems part of the Chapter 19 on "Cardiac Output Monitoring."

3. Mixed venous oxygen saturation (SvO_2)

It is a measurement of oxygen saturation

from a blood sample from the pulmonary artery through a PAC. SvO_2 is the oxygen content of the blood that returns to the heart and is determined by cardiac output, hemoglobin concentration, oxygen supply (ventilatory settings, fraction of inspired oxygen, etc.), and tissue oxygen consumption [90].

Important causes of low SvO_2 are low or inadequate cardiac output, anemia, hypoxemia, or increased O_2 demand (sepsis, hyperthermia, burns, seizures, or shivering). The normal value of SvO_2 is 60–80%, and SvO_2 less than 65% is of poor prognostic value [93]. SvO_2 below 60% poses a serious risk of tissue hypoxia and needs urgent corrective measures.

Use of PAC in clinical practice. When and why?

The use of the PAC has decreased substantially and is not used routinely in most critically ill patients because:

1. PAC is a highly invasive technique with several complications. With the availability of less invasive hemodynamic monitoring techniques, the use of the PAC has declined in the last three decades [57, 94–96].
2. PAC failed to improve the outcome and survival [97–101].
3. Measurements of cardiac output by PAC are frequently inaccurate in critically ill patients [102, 103].
4. Static parameter like pulmonary artery occlusion pressure does not predict fluid volume [104, 105] or fluid responsiveness and therefore is not useful in fluid management in critically ill patients [106–108].

Indications

The unique feature of PAC monitoring is that it can directly measure the pressures in the right heart and pulmonary circulation [109]. PAC is the only device that efficiently assesses and continuously monitors the right ventricle function [110].

Currently, the use of PAC must be restricted to very few selected critically ill patients and complex clinical situations [92]. The most frequent indications are [2, 64, 111–113]:

- To differentiate the causes of various unexplained or multi-factorial shock states. PAC helps to differentiate cardiogenic and non-cardiogenic causes of severe shock.
- To evaluate right ventricular heart failure, pulmonary edema, pulmonary hypertension, or refractory shock, and helps in the planning of more precise fluid, inotropes, vasodilators, and diuretics treatment.
- Perioperatively in high-risk patients with severe pulmonary hypertension and acute right ventricular failure, PAC helps in the proper administration of fluids and vasopressors.
- To diagnose cardiac tamponade or constrictive pericarditis when clinical and echocardiographic findings are not conclusive.
- Preoperative assessment of intracardiac shunt, congenital heart disease, or right-sided valvular disease.

Complications

The common complications which may occur from the use of the PAC are cardiac arrhythmias, pulmonary artery rupture or thrombosis, pulmonary hemorrhage, balloon rupture, pneumothorax, catheter malposition or intra-cardiac knotting, tricuspid or pulmonary valve injury, right atrial thrombosis, internal jugular/subclavian vein stenosis, venous thromboembolism, electromechanical dissociation, and right-sided endocarditis and catheter-related bloodstream infection.

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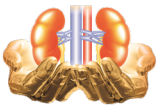
KidneyEducation 

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