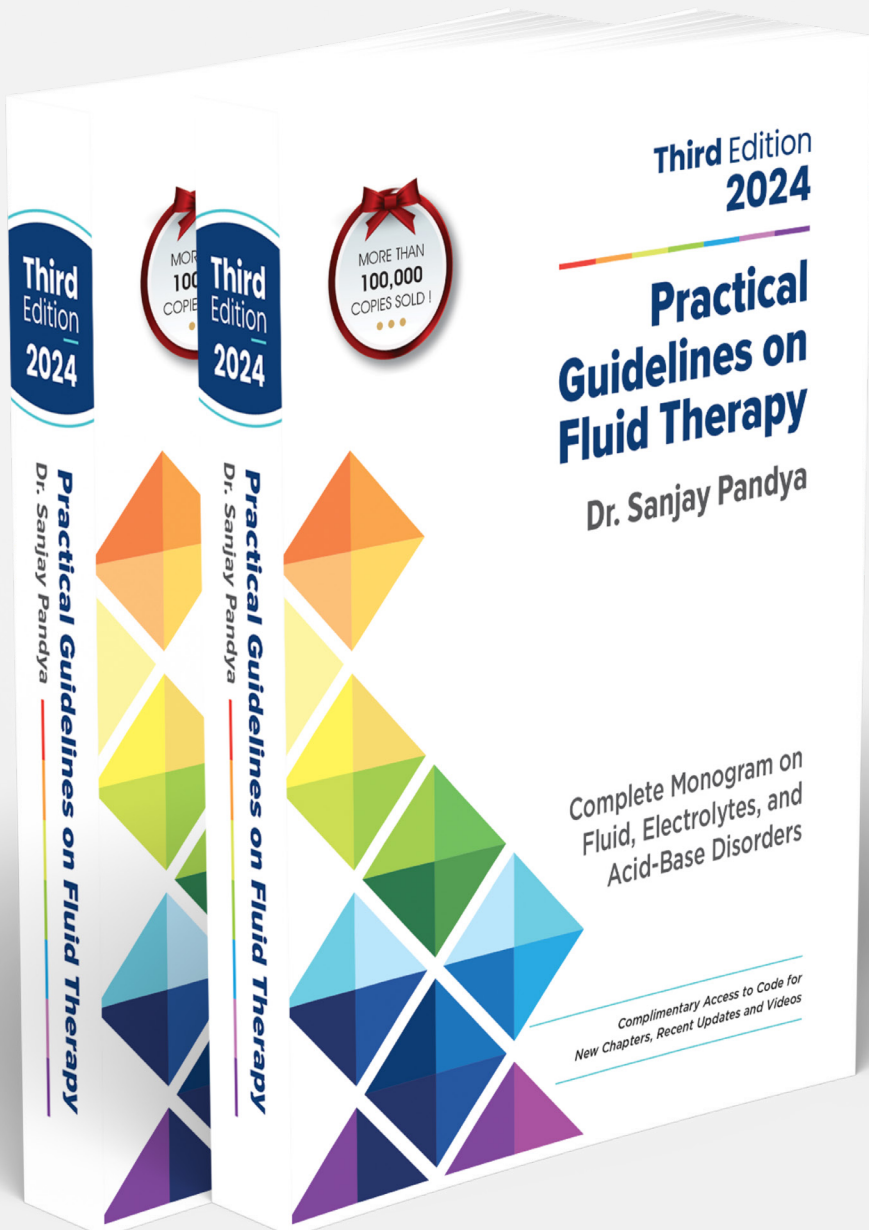




Fluid
therapy.org

Chapter 17:

Static Hemodynamic Monitoring Techniques



To get a copy of the book, visit: www.fluidtherapy.org

17

Static Hemodynamic Monitoring Techniques

Inferior Vena Cava Assessment	175	Arterial Cannulation	181
Physiological principles	176	Use and technique	181
IVC in spontaneous respiration..	176	Indications and contraindications..	182
IVC in patients on ventilator	176	Radial or femoral, which artery?...	182
Measurement techniques	177	Pulmonary Artery Catheter	
Maximal IVC diameter	177	Monitoring	183
In spontaneous breathing.....	177	Description and technique.....	183
In mechanically ventilated		What information does PAC	
patients.....	177	provide?.....	183
IVC diameter variation.....	178	Pulmonary artery occlusion	
In mechanical ventilation.....	178	pressure	184
In spontaneous breathing.....	179	Cardiac output.....	184
Central Venous Pressure		Mixed venous oxygen	
Monitoring	179	saturation.....	184
Factors determining CVP	179	PAC - when and why?	185
Interpretation of the value of CVP..	179	Indications	185
The use CVP in clinical practice ...	180	Complications.....	185

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.

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].

IVC Distensibility Index

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

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 ≥ 8 mL/kg with positive end-expiratory pressure (PEEP) ≤ 5 cm H₂O, 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 H₂O [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 (Δ IVC) 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 cmH₂O increase in PEEP will cause a 2.5 cmH₂O 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 H₂O (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 judiciously 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 bedside 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 (SvO₂).

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 (SvO₂)

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.

REFERENCES

1. Levitov A, Frankel HL, Blaivas M, et al. Guidelines for the appropriate use of bedside general and cardiac ultrasonography in the evaluation of critically ill patients - Part II: Cardiac ultrasonography. *Crit Care Med.* 2016;44(6):1206–27.
2. Cecconi M, De Backer D, Antonelli M, et al. Consensus on circulatory shock and hemodynamic monitoring. Task force of the European Society of Intensive Care Medicine. *Intensive Care Med.* 2014;40(12):1795–815.
3. Schmidt GA. POINT: Should acute fluid resuscitation be guided primarily by inferior vena cava ultrasound for patients in shock? *Yes Chest.* 2017;151(3):531–532.
4. Charbonneau H, Riu B, Faron M, et al. Predicting preload responsiveness using simultaneous recordings of inferior and superior vena cavae diameters. *Crit Care.* 2014;18(5):473.
5. Vignon P, Repesse X, Begot E, et al. Comparison of echocardiographic indices used to predict fluid responsiveness in ventilated patients. *Am J Respir Crit Care Med.* 2017;195(8):1022–1032.
6. Miller A, Mandeville J. Predicting and measuring fluid responsiveness with echocardiography. *Echo Res Pract.* 2016;3(2):G1–G12.
7. Muller L, Bobbia X, Toumi M, et al. Respiratory variations of inferior vena cava diameter to predict fluid responsiveness in spontaneously breathing patients with acute circulatory failure: need for a cautious use. *Crit Care.* 2012;16(5):R188.
8. Yanagawa Y, Sakamoto T, Okada Y. Hypovolemic shock evaluated by sonographic measurement of inferior vena cava during resuscitation in trauma patients. *J Trauma.* 2007;63(6):1245–8.
9. Airapetian N, Maizel J, Alyamani O, et al. Does inferior vena cava respiratory variability predict fluid responsiveness in spontaneously breathing patients? *Crit Care.* 2015;19:400.
10. Vieillard-Baron A, Evrard B, Repesse X, et al. Limited value of end-expiratory inferior vena cava diameter to predict fluid responsiveness impact of intra-abdominal pressure. *Intensive Care Med.* 2018;44(2):197–203.
11. Marx G, Schindler AW, Mosch C, et al. Intravascular volume therapy in adults: guidelines from the Association of the Scientific Medical Societies in Germany. *Eur J Anesthesiol.* 2016;33(7):488–521.
12. Yanagawa Y, Nishi K, Sakamoto T, et al. Early diagnosis of hypovolemic shock by sonographic measurement of inferior vena cava in trauma patients. *J Trauma.* 2005;58(4):825–9.
13. Dipti A, Soucy Z, Surana A, et al. Role of inferior vena cava diameter in assessment of volume status: a meta-analysis. *Am J Emerg Med.* 2012;30(8):1414–1419.e1.
14. Furtado S, Reis L. Inferior vena cava evaluation in fluid therapy decision making in intensive care: practical implications. *Rev. bras. ter. intensiva [online].* 2019;31(2):240–247.
15. Lee CW, Kory PD, Arntfield RT. Development of a fluid resuscitation protocol using inferior vena cava and lung ultrasound. *J Crit Care.* 2016;31(1):96–100.
16. Jardin F, Vieillard-Baron A. Ultrasonographic examination of the venae cavae. *Intensive Care Med.* 2006;32(2):203–206.
17. Millington SJ. Ultrasound assessment of the inferior vena cava for fluid responsiveness: easy, fun, but unlikely to be helpful *Can J Anesth/J Can Anesth* 2019;66(6):633–638.
18. Si X, Xu H, Liu Z, et al. Does respiratory variation in inferior vena cava diameter predict fluid responsiveness in mechanically ventilated patients? A systematic review and meta-analysis. *Anesth Analg.* 2018;127(5):1157–1164.
19. De Backer D, Fagnoul D. Intensive Care Ultrasound: VI. Fluid Responsiveness and Shock Assessment. *Am Thorac Soc.* 2014;11(1):129–36.
20. Zhang Z, Xu X, Ye S, et al. Ultrasonographic measurement of the respiratory variation in the inferior vena cava diameter is predictive of fluid responsiveness in critically ill patients: systematic review and meta-analysis. *Ultrasound Med Biol.* 2014;40(5):845–53.
21. Long E, Oakley E, Duke T, et al. Paediatric Research in Emergency Departments International Collaborative (PREDICT). Does respiratory variation in inferior vena cava diameter predict fluid responsiveness: A systematic review and meta-analysis. *Shock* 2017;47(5):550–559.
22. Kory P. COUNTERPOINT: should acute fluid resuscitation be guided primarily by inferior vena cava ultrasound for patients in shock? *No. Chest.* 2017;151(3):533–536.
23. Schmidt GA. Rebuttal from Dr Schmidt. *Chest.* 2017;151(3):536–537.
24. Kory P. Rebuttal from Dr Kory. *Chest.* 2017;151(3):537–538.
25. Barbier C, Loubieres Y, Schmit C, et al. Respiratory changes in inferior vena cava diameter are helpful in predicting fluid responsiveness in ventilated septic patients. *Intensive Care Med* 2004;30(9):1740–6.
26. Feissel M, Michard F, Faller JP, et al. The respiratory variation in inferior vena cava diameter as a guide to fluid therapy. *Intensive Care Med* 2004;30(9):1834–1837.
27. Moretti R, Pizzi B. Inferior vena cava distensibility as a predictor of fluid responsiveness in patients with subarachnoid hemorrhage. *Neurocrit Care.* 2010;13(1):3–9.
28. Machare-Delgado E, Decaro M, Marik PE. Inferior vena cava variation compared to pulse contour analysis as predictors of fluid responsiveness: a prospective cohort study. *J Intensive Care Med* 2011;26(2):116–24.
29. Mandeville JC, Colebourn CL. Can transthoracic echocardiography be used to predict fluid responsiveness in the critically ill patient? A systematic review. *Crit Care Res Pract* 2012;2012:513480.
30. Lu N, Xi X, Jiang L, et al. Exploring the best predictors of fluid responsiveness in patients with septic shock. *Am J Emerg Med* 2017;35(9):1258–1261.

31. Huang H, Shen Q, Liu Y, et al. Value of variation index of inferior vena cava diameter in predicting fluid responsiveness in patients with circulatory shock receiving mechanical ventilation: a systematic review and meta-analysis. *Crit Care* 2018;22(1):204.
32. Mohammad AWM, Saad-elden ES, Mohammad EK, et al. Distensibility index of inferior vena cava and pulse pressure variation as predictors of fluid responsiveness in mechanically ventilated shocked patients. *Journal of Emergency Medicine, Trauma & Acute Care* 2020;2020(1).
33. Sobczyk D, Nycz K, Andruszkiewicz P, et al. Ultrasonographic caval indices do not significantly contribute to predicting fluid responsiveness immediately after coronary artery bypass grafting when compared to passive leg raising. *Cardiovasc Ultrasound* 2016;14(1):23.
34. Zhang H, Zhang Q, Chen X, et al. Respiratory variations of inferior vena cava fail to predict fluid responsiveness in mechanically ventilated patients with isolated left ventricular dysfunction. *Ann. Intensive Care* 2019;9(1):113.
35. Orso D, Paoli I, Piani T, et al. Accuracy of ultrasonographic measurements of inferior vena cava to determine fluid responsiveness: a systematic review and meta-analysis. *J Intensive Care Med*. 2020;35(4):354–363.
36. Sarıtaş A, Zincircioğlu Ç, Uzun SP, et al. Comparison of inferior vena cava collapsibility, distensibility, and delta indices at different positive pressure supports and prediction values of indices for intravascular volume status. *Turk J Med Sci*. 2019;49(4):1170–1178.
37. Farcy DA, Jain A, Dalley M, et al. Pitfalls in using central venous pressure as a marker of fluid responsiveness. *Emerg Med* 2016;48(1):18–28.
38. Vieillard-Baron A, Chergui K, Rabiller A, et al. Superior vena caval collapsibility as a gauge of volume status in ventilated septic patients. *Intensive Care Med* 2004;30(9):1734–1739.
39. Lanspa MJ, Grissom CK, Hirshberg EL, et al. Applying dynamic parameters to predict hemodynamic response to volume expansion in spontaneously breathing patients with septic shock. *Shock*. 2013;39(2):155–60.
40. Preau S, Bortolotti P, Colling D, et al. Diagnostic accuracy of the inferior vena cava collapsibility to predict fluid responsiveness in spontaneously breathing patients with sepsis and acute circulatory failure. *Crit Care Med*. 2017;45(3):e290–e297.
41. Corl KA, George NR, Romanoff J, et al. Inferior vena cava collapsibility detects fluid responsiveness among spontaneously breathing critically-ill patients. *J Crit Care*. 2017;41:130–137.
42. Bortolotti P. Inferior vena cava respiratory variations. A useful tool at bedside to guide fluid therapy in spontaneously breathing patients. *SHOCK* 2018;49(2):235–236.
43. Bortolotti P, Colling D, Colas V, et al. Respiratory changes of the inferior vena cava diameter predict fluid responsiveness in spontaneously breathing patients with cardiac arrhythmias. *Ann. Intensive Care* 2018;8(1):79.
44. Szabó M, Bozó A, Darvas K, et al. Role of inferior vena cava collapsibility index in the prediction of hypotension associated with general anesthesia: an observational study. *BMC Anesthesiol* 2019;19:139.
45. Williams K, Ablordeppey E, Theodoro D, et al. The diagnostic accuracy of inferior vena cava collapsibility versus passive leg raise testing in determining volume responsiveness in emergency department patients with shock. *Proceedings of the 40th Critical Care Congress, Society of Critical Care Congress*. *Crit Care Med* 2011;39:8.
46. Corl K, Napoli AM, Gardiner F. Bedside sonographic measurement of the inferior vena cava caval index is a poor predictor of fluid responsiveness in emergency department patients. *Emerg Med Australas*. 2012;24(5):534–539.
47. Juhl-Olsen P, Vistisen ST, Christiansen LK, et al. Ultrasound of the inferior vena cava does not predict hemodynamic response to early hemorrhage. *J Emerg Med* 2013;45(4):592–597.
48. De Valk S, Olgers TJ, Holman M, et al. The caval index: an adequate non-invasive ultrasound parameter to predict fluid responsiveness in the emergency department? *BMC Anesthesiology*. 2014;14:114.
49. Block J, Mackenzie D. Fluid Responsiveness in a Hemodynamically Unstable Patient. *EMresident15 Dec 2018 Visit: <https://www.emra.org/emresident/article/fluid-responsiveness/>*.
50. McIntyre LA, Hébert PC, Fergusson D, et al. Canadian Critical Care Trials Group. A survey of Canadian intensivists' resuscitation practices in early septic shock. *Crit Care*. 2007;11(4):R74.
51. Cannesson M, Pestel G, Ricks C, et al. Hemodynamic monitoring and management in patients undergoing high risk surgery: a survey among North American and European anesthesiologists. *Crit Care*. 2011;15(4):R197.
52. Gelman S. Venous function and central venous pressure: a physiologic story. *Anesthesiology*. 2008;108(4):735–48.
53. Shojaee M, Sabzghabaei A, Alimohammadi H, et al. Effect of positive end-expiratory pressure on central venous pressure in patients under mechanical ventilation. *Emerg (Tehran)*. 2017;5(1):e1.
54. Cecconi M, Hofer C, Teboul JL, et al. Fluid challenges in intensive care: the FENICE study: a global inception cohort study. *Intensive Care Med* 2015;41(9):1529–37.
55. Marik PE, Baram M, Vahid B. Does the central venous pressure predict fluid responsiveness? A systematic review of the literature and the tale of seven mares. *Chest*. 2008;134(1):172–8.
56. Marik PE, Cavallazzi R. Does the Central Venous Pressure (CVP) predict fluid responsiveness: An update meta-analysis and a plea for some common sense. *Crit Care Med*. 2013;41(7):1774–81.
57. Martin ND, Codner P, Greene W, et al. Contemporary hemodynamic monitoring, fluid responsiveness, volume optimization, and endpoints of resuscitation:

- an AAST critical care committee clinical consensus. *Trauma Surg Acute Care Open* 2020;5(1):e000411.
58. Chen C, Zhou Y, Wang P, et al. Elevated central venous pressure is associated with increased mortality and acute kidney injury in critically ill patients: a meta-analysis. *Crit Care* 2020;24(1):80.
 59. Magder S. Value of CVP: an epidemiological or physiological question? *Intensive Care Med* 2016;42(3):458–459.
 60. Eskesen TG, Wetterslev M, Perner A. Systematic review including re-analyses of 1148 individual data sets of central venous pressure as a predictor of fluid responsiveness. *Intensive Care Med*. 2016;42(3):324–332.
 61. Biais M, Ehrmann S, Mari A, et al. Clinical relevance of pulse pressure variations for predicting fluid responsiveness in mechanically ventilated intensive care unit patients: the grey zone approach. *Crit Care*. 2014;18(6):587.
 62. De Backer D, Vincent J. Should we measure the central venous pressure to guide fluid management? Ten answers to 10 questions. *Crit Care* 2018;22(1):43.
 63. Rivers E, Nguyen B, Havstadt S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med*. 2001;345(19):1368–77.
 64. Teboul JL, Saugel B, Cecconi M, et al. Less invasive hemodynamic monitoring in critically ill patients. *Intensive Care Med*. 2016;42(9):1350–1359.
 65. Su L, Pan P, Li D, et al. Central Venous Pressure (CVP) reduction associated with higher Cardiac Output (CO) favors good prognosis of circulatory shock: a single-center, retrospective cohort study. *Front Med (Lausanne)*. 2019;6:216.
 66. Pinsky MR, Kellum JA, Bellomo R. Central venous pressure is a stopping rule, not a target of fluid resuscitation. *Crit Care Resusc* 2014;16(4):245–246.
 67. Boyd JH, Forbes J, Nakada TA, et al. Fluid resuscitation in septic shock: a positive fluid balance and elevated central venous pressure are associated with increased mortality. *Crit Care Med*. 2011;39(2):259–65.
 68. Marik PE. Iatrogenic salt water drowning and the hazards of a high central venous pressure. *Ann Intensive Care*. 2014;4:21.
 69. Lee J, De Louw E, Niemi M, et al. Association between fluid balance and survival in critically ill patients. *J Intern Med*. 2015;277(4):468–77.
 70. Sondergaard S, Parkin G, Aneman A. Central venous pressure: soon an outcome-associated matter. *Curr Opin Anaesthesiol*. 2016;29(2):179–85.
 71. Li D, Wang X, Liu D. Association between elevated central venous pressure and outcomes in critically ill patients. *Ann. Intensive Care* 2017;7(1):83.
 72. Mullens W, Abrahams Z, Francis GS, et al. Importance of venous congestion for worsening of renal function in advanced decompensated heart failure. *J Am Coll Cardiol*. 2009;53(7):589–96.
 73. Sen S, Chini EN, Michael J, et al. Complications after unintentional intra-arterial injection of drugs: risks, outcomes, and management strategies. *Mayo Clin Proc*. 2005;80(6):783–795.
 74. Brzezinski M, Luisetti T, London MJ. Radial artery cannulation: a comprehensive review of recent anatomic and physiologic investigations. *Anesth Analg*. 2009;109(6):1763–81.
 75. O’Grady NP, Alexander M, Burns LA, et al. Summary of recommendations: Guidelines for the Prevention of Intravascular Catheter-related Infections. *Clin Infect Dis*. 2011;52(9):1087–99.
 76. Miller AG, Bardin AJ. Review of ultrasound-guided radial artery catheter placement. *Respir Care*. 2016;61(3):383–8.
 77. Scheer B, Perel A, Pfeiffer UJ. Clinical review: complications and risk factors of peripheral arterial catheters used for hemodynamic monitoring in anaesthesia and intensive care medicine. *Crit Care*. 2002;6(3):199–204.
 78. Camporota L, Beale R. Pitfalls in hemodynamic monitoring based on the arterial pressure waveform. *Crit Care* 2010;14(2):124.
 79. Hatib F, Jansen JR, Pinsky MR. Peripheral vascular decoupling in porcine endotoxemic shock. *J Appl Physiol*. 2011;111(3):853–60.
 80. Farkas J. PulmCrit: A-lines in septic shock: the wrist versus the groin. Aug 2018 Visit: <https://emcrit.org/pulmcrit/a-line/>.
 81. Koyuncu O, Leung S, Asha SA, et al. The present and future of indwelling arterial catheter: An anesthesiologist’s perspective. *Clin Res Trials*. 2016;2(2):157–158.
 82. Galluccio ST, Chapman MJ, Finnis ME. Femoral-radial arterial pressure gradients in critically ill patients. *Crit Care Resusc*. 2009;11(1):34–38.
 83. Kim W, Jun J, Huh J, et al. Radial to femoral arterial blood pressure differences in septic shock patients receiving high-dose norepinephrine therapy. *Shock*. 2013;40(6):527–531.
 84. Fuda G, Denault A, Deschamps A, et al. Risk factors involved in central-to-radial arterial pressure gradient during cardiac surgery. *Anesth Analg*. 2016;122(3):624–632.
 85. Greer M, Carney S, McPheeters R, et al. Radial arterial lines have a higher failure rate than femoral. *West J Emerg Med*. 2018;19(2):364–371.
 86. Lakhali K, Robert-Edan V. Invasive monitoring of blood pressure: a radiant future for brachial artery as an alternative to radial artery catheterisation? *J Thorac Dis*. 2017;9(12):4812–4816.
 87. Lorente L, Santacreu R, Martín MM, et al. Arterial catheter-related infection of 2,949 catheters. *Crit Care* 2006;10(3):R83.
 88. O’Horo JC, Maki DG, Krupp AE, et al. Arterial catheters as a source of bloodstream infection: a systematic review and meta-analysis. *Crit Care Med* 2014;42(6):1334–9.
 89. Kalantari K, Chang JN, Ronco C, et al. Assessment of intravascular volume status and volume responsiveness in critically ill patients. *Kidney Int*. 2013;83(6):1017–28.
 90. Lee CP, Bora V. Anesthesia monitoring of mixed venous saturation. *StatPearls* [Internet]. Feb 2020 Visit: <https://www.ncbi.nlm.nih.gov/books/NBK539835/>.

91. Nickson C. Pulmonary Artery Catheter. Life in the Fast Lane CCC. Sept 2019 Visit: <https://litfl.com/pulmonary-artery-catheter/>.
92. Demiselle J, Mercat A, Asfar P. Is there still a place for the Swan–Ganz catheter? Yes. *Intensive Care Med* 2018;44(6):954–956.
93. Hartog C, Bloos F. Venous oxygen saturation. *Best Pract Res Clin Anaesthesiol*. 2014;28(4):419–28.
94. Litton E, Morgan M. The PiCCO monitor: a review. *Anaesthesia and intensive care*. 2012;40(3):393–409.
95. Seifi A, Elliott RJ, Elsehety MA. Usage of Swan-Ganz catheterization during the past 2 decades in United States. *J Crit Care* 2016;35:213–4.
96. De Backer D, Vincent JL. The pulmonary artery catheter: is it still alive? *Current Opinion in Critical Care* 2018;24(3):204–208.
97. Richard C, Warszawski J, Anguel N, et al. Early use of the pulmonary artery catheter and outcomes in patients with shock and acute respiratory distress syndrome: a randomized controlled trial. *Journal of the American Medical Association* 2003;290(20):2713–20.
98. Harvey S, Harrison DA, Singer M, et al. Assessment of the clinical effectiveness of pulmonary artery catheters in management of patients in intensive care (PAC-Man): a randomised controlled trial. *Lancet*. 2005;366(9484):472–7.
99. National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network, Wheeler AP, et al. Pulmonary-artery versus central venous catheter to guide treatment of acute lung injury. *N Engl J Med*. 2006;354(21):2213–24.
100. Harvey S, Young D, Brampton W, et al. Pulmonary artery catheters for adult patients in intensive care. *Cochrane Database Syst Rev*. 2006;(3):CD003408.
101. Rajaram SS, Desai NK, Kalra A, et al. Pulmonary artery catheters for adult patients in intensive care. *Cochrane Database of Systematic Reviews* 2013;2013(2):CD003408.
102. Phillips RA, Hood SG, Jacobson BM, et al. Pulmonary artery catheter (PAC) accuracy and efficacy compared with flow probe and transcutaneous Doppler (USCOM): an ovine cardiac output validation. *Crit Care Res Pract* 2012;2012:621494.
103. Marik PE. Obituary: pulmonary artery catheter 1970 to 2013. *Ann Intensive Care*. 2013;3(1):38.
104. Kumar A, Anel R, Bunnell E, et al. Pulmonary artery occlusion pressure and central venous pressure fail to predict ventricular filling volume, cardiac performance, or the response to volume infusion in normal subjects. *Crit Care Med*. 2004;32(3):691–9.
105. Oohashi S, Endoh H, Oohashi S, et al. Does central venous pressure or pulmonary capillary wedge pressure reflect the status of circulating blood volume in patients after extended transthoracic esophagectomy? *J Anesth* 2005;19(1):21–25.
106. Keller G, Sinavsky K, Desebbe O, et al. Combination of continuous pulse pressure variation monitoring and cardiac filling pressure to predict fluid responsiveness. *J Clin Monit Comput*. 2012;26(6):401–5.
107. Michard F, Teboul JL. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. *Chest*. 2002;121(6):2000–2008.
108. Osman D, Ridel C, Ray P, et al. Cardiac filling pressures are not appropriate to predict hemodynamic response to volume challenge. *Crit Care Med* 2007;35(1):64–68.
109. Huygh J, Peeters Y, Bernards J, et al. Hemodynamic monitoring in the critically ill: an overview of current cardiac output monitoring methods. *F1000Res*. 2016;5:F1000.
110. Ventetuolo CE, Klinger JR. Management of acute right ventricular failure in the intensive care unit. *Ann Am Thorac Soc* 2014;11(5):811–822.
111. Chatterjee K. The Swan-Ganz catheters: past, present, and future. A viewpoint. *Circulation*. 2009;119(1):147–52.
112. Rhodes A, Evans LE, Alhazzani W, et al. Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock: 2016. *Intensive Care Med*. 2017;43(3):304–377.
113. Ziccardi MR, Khalid N. Pulmonary Artery Catheterization. *StatPearls* [Internet]. Feb 2020 (Visit: <https://www.ncbi.nlm.nih.gov/books/NBK482170/>).

KidneyEducation

Join the Mission to Fight Kidney Diseases

Explore the world's largest multilingual website created by a global team of over 100 nephrologists.

www.KidneyEducation.com

- » Read online or download the 200-page book "Save Your Kidneys" in 40 languages—completely free.
- » This comprehensive resource offers valuable information on preventing and managing common kidney problems, tailored for kidney patients and their families.
- » It's an authentic guide, prepared by nephrologists and free from any external funding.