12 Invasive hemodynamic monitoring in obstetrics

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CENTRAL VENOUS ACCESS

Approximately 0.17% to 1.1% of all pregnant patients require admission to the intensive care unit (ICU) at some point during their pregnancies (1). Obstetric complications, including hemorrhage and hypertensive disorders (primarily pre-eclampsia and eclampsia), account for most of these ICU admissions comprising 47% to 93% of patients (2). Understanding the main hemodynamic changes associated with pregnancy is crucial when invasive monitoring is applied to the critically ill pregnant patients (Table 1). Central venous access is more commonly obtained through internal jugular (IJ) vein or subclavian vein catheterization, as seen in the nonpregnant population as well.

The main indications for central venous catheter (CVC) placement include administration of vasoactive medications such as norepinephrine or dopamine, infusion of caustic or hypertonic solutions such as potassium chloride or parenteral alimentation, administration of incompatible medicines through a multi-lumen catheter, requirement of acute or subacute renal replacement therapy, necessity of hemodynamic monitoring or transvenous cardiac pacing, and rapid infusion of fluids (3,4). Due to the emergent nature of most CVC placements and their lifesaving indications, there are truly no absolute contraindications to their placement. A few general contraindications do exist and include infection of the area overlying the target vein, thrombosis of the target vein, and coagulopathy (4).

Recent statistics show that approximately 5 million CVCs are placed each year in the United States with one or more complications occurring in >15% of catheter placements (5,6). The risk of complications increases with operator inexperience, emergent placement, patient obesity, history of difficult cannulation, and coagulopathy. Common mechanical complications include arterial puncture, hematoma formation, pneumothorax, hemothorax, arrhythmias, and improper location of the catheter (7,8). Venous thrombosis is another complication that occurs less frequently in the subclavian vein and more frequently with cannulation of the femoral vein (5,6). The risk of pneumothorax during CVC insertion increases with hyperinflation of the lungs associated with mechanical ventilation with large tidal volumes or with increased positive end-expiratory pressure. Inadvertent arterial puncture can usually be distinguished by the pulsatile nature of the flow and the red color of arterial blood (4). If in doubt, the operator may insert a small 18-gauge catheter over the guidewire and determine the waveform of the vessel to distinguish arterial from venous cannulation (4). Alternatively, the 18-gauge catheter may be connected to a regular intravenous tubing that is allowed to fill with blood, and when raised, the column of blood should drop if the catheter is inside a vein. Finally, a chest x-ray to confirm proper placement should be obtained

after central venous catheterization regardless of insertion site (except for the femoral vein placement).

In addition to mechanical complications, catheter-related infections pose a significant risk to the patient. Infection of the CVC may occur locally at the insertion site, from hub colonization and subsequent infection through the catheter lumen, or through hematogenous seeding of the catheter (5). The Centers for Disease Control and Prevention recommend the subclavian vein as the site of choice to reduce the risk of catheter-related sepsis (9). The Institute for Healthcare Improvement recommends five steps to reduce central-line infections: hand washing, maximal barrier precautions, chlorhexidine skin antisepsis, selection of an optimal catheter site, and prompt removal of the CVC when no longer needed. Antiseptic-containing hubs (chlorhexidine) and antimicrobial-impregnated catheters (minocycline and rifampin) have been shown to decrease the rate of catheter-related bloodstream infections when compared with non-impregnated catheters and should be considered in all CVC insertions when the institutional infection rate exceeds 2% (4,5).

The use of ultrasound guidance has been shown in various prospective/randomized trials and two meta-analyses since 1990 to reduce mechanical complications of CVC insertion into the IJ and improve first-pass success (10). Two-dimensional ultrasound can be used indirectly or directly. With indirect ultrasound guidance, the ultrasound is used for vessel localization, and the puncture site is identified. After this, puncture of the vein is performed without real-time ultrasound guidance. Direct ultrasound guidance allows visualization of the needle puncture in real time after localization of the vessel and can employ one or two operators.

In 2003, Hind et al. evaluated data from 18 studies in a meta-analysis that excluded Doppler studies (11). They found that two-dimensional ultrasound was significantly better for central venous access when compared with the traditional landmark methods and was more effective in avoiding failures in the first attempt of venous cannulation.

Particularly for IJ catheter placements, ultrasound guidance is now considered the standard of care.

Internal Jugular Vein

IJ cannulation has a lower risk of pneumothorax as compared with the subclavian approach. However, the risks of infection and thrombosis are higher with the IJ.

The anatomical landmarks commonly used to place a central line in the IJ consist of the triangle formed by the clavicle and the sternal and clavicular heads of the sternocleidomastoid muscle. The apex of this triangle corresponds to the separation of the two heads of the muscle. In the apex, the carotid pulse is felt, and the artery is gently retracted to the

Table 1 Hemodynamic Changes in Pregnancy

	Nonpregnant	Pregnant
Cardiac output (L/min)	4.3 ± 0.9	6.2 ± 1.0
Heart rate (beats/min)	71 ± 10.0	83 ± 10.0
Stroke volume (mL)	65	75–85
Systemic vascular resistance (dynes × s × cm ⁻⁵)	1530 ± 520	1210 ± 266
Pulmonary vascular resistance (dynes × s × cm ⁻⁵)	119 ± 47.0	78 ± 22.0
Colloid oncotic pressure (mmHg)	14.5 ± 2.5	10.5 ± 2.7
Mean arterial pressure (mmHg)	86.4 ± 7.5	90.3 ± 5.8
Pulmonary capillary wedge pressure (mmHg)	6.3 ± 2.1	7.5 ± 1.8
Central venous pressure (mmHg)	3.7 ± 2.6	3.6 ± 2.5
Left ventricular stroke work index (g × min × m ²)	41 ± 8	48 ± 6
Blood volume (L)	4.5	6.5
Plasma volume (L)	2.5	3.5–4.6

midline (the IJ usually is located lateral to the carotid pulse). The needle should be inserted at the apex level just lateral to the carotid pulse. The site is prepared and draped in a sterile fashion. The operator is gowned and gloved for gaining venous access and for positioning the catheter. The patient is positioned, and the head is tilted to the side opposite to the vessel selected for cannulation. The Trendelenburg position aids in distending the venous caliber. With the use of 1% xylocaine and a 21- or 22-gauge 2-inch needle, the skin is injected and a wheal is made over the planned cannulation site. The needle is then advanced with the bevel up at a 45° angle toward the ipsilateral nipple. Turning the patient's head to the opposite side will straighten the course of the vein and thus facilitate the procedure. The right side is usually preferred since it has a straighter course toward the superior vena cava. In most cases, the vein will be found at a depth of less than 2 cm. Often the vein walls will collapse against each other by the pressure of the needle, and the vessel is completely penetrated without blood return. Slow retraction of the needle while maintaining negative pressure on the syringe will overcome this problem, as during retraction, the needle will re-enter the vessel and blood return will be obtained. Once the vessel is located with this "finding needle," an 18-gauge needle is used to puncture the vein following the tract established with the initial small needle. After this, the syringe is detached from the needle (which should stay in place), and a guidewire is advanced through it. After the wire is passed into the vessel, the needle is withdrawn over the wire. A scalpel is used to make a 5-mm cut in the skin, following which the larger venous dilator is inserted to dilate the tract. After this, the dilator is removed, and the catheter is advanced over the guidewire. The wire is then withdrawn, and the catheter remains in place. This same principle (Seldinger) is applied for the placement of lines in the subclavian or femoral veins.

When using ultrasound guidance for IJ catheter insertion, blood is hypoechoic and appears dark on the ultrasound screen with the IJ typically seen anterior and lateral to the carotid artery. The IJ vein can be distinguished from the artery because it is compressible, nonpulsatile, and distended by Trendelenburg position or the Valsalva maneuver (10). There are several factors that must be considered when using the ultrasound for IJ catheter insertion: (i) excessive pressure applied to the transducer decreases IJ vein diameter, making it harder to access; (ii) extreme contralateral head rotation can

decrease IJ vein diameter and increase overlap onto the carotid artery; (iii) employing the Trendelenburg position and using correct head positioning can significantly increase IJ vein diameter and facilitate placement; and (iv) patient intravascular volume status can affect the size of the IJ.

Passage of a needle into the IJ can be done with either a transverse (short axis) view or a longitudinal (long axis) view. The transverse view is associated with a shorter learning curve and makes it easier to visualize smaller vessels (10). Using the longitudinal (long axis) view, as recommended by the American College of Emergency Physicians, allows better visualization of the advancing needle tip, which may reduce perforation of the posterior wall of the vessel and potential entry into the artery (10).

Figure 1 illustrates the placement of a CVC in the IJ site.

Subclavian Vein

Due to the low risk of thrombosis and infection, in addition to the patient's comfort and easy access, the subclavian vein is the preferred site for CVC placement in the ICU setting.

Coagulopathy is a relative contraindication since in the event of bleeding, the vessels (subclavian vein or artery) are not directly accessible for compression.

The subclavian vein lies just beneath the clavicle. Exposure of the vein may be improved by placing a small rolled towel between the shoulder blades. The right side is also preferred since the right lung apex is lower than the left, diminishing the chances of a pneumothorax when this side is chosen. Also injuries to the thoracic duct may happen (albeit infrequently) when attempting to access the left subclavian vein.

The head of the patient is turned to the opposite side, and Trendelenburg positioning is accomplished. The entry site is usually 2 to 3 cm caudal to the midpoint of the clavicle. The 18-gauge introducer needle is advanced with the bevel up toward the sternal notch and beneath the clavicle. Care should be taken to avoid directing the needle too caudally (risk of pneumothorax) or too cephalad (risk of subclavian artery puncture). Once blood flow is obtained, the bevel should be rotated 90° clockwise so that the wire, when advanced, will follow the direction toward the superior vena cava. The Seldinger technique is then followed to advance the catheter.

When considering the subclavian vein, several recent articles have suggested that ultrasound can increase the likelihood of successful placement of a subclavian catheter

Figure 1 Placement of a central venous catheter in the right internal jugular vein. Source: With permission from New England Journal of Medicine.

despite the presence of the clavicle (12). In addition, Doppler can also be used to distinguish artery from vein due to the difficulty in identifying the vein by compression alone (13).

Figure 2 illustrates the placement of a CVC in the subclavian site.

Femoral Vein

Placement of the femoral catheter begins with optimal exposure of the femoral region by externally rotating and abducting the leg away from the midline. The femoral vein is 1 to 2 cm medial to the artery and runs distal to the inguinal ligament. The vein can be located by palpating the pulsating femoral artery. The site must be prepped and draped and the insertion site must be anesthetized with local anesthesia. The introducer needle should then be inserted at a 45° angle directed along the course of the vein 2 cm below the inguinal ligament (2 cm below an imaginary line that connects the symphysis pubis and the superior–anterior iliac crest). Avoid puncturing above the inguinal ligament since retroperitoneal hemorrhage may ensue. The femoral artery should be palpated at all times while introducing and advancing the needle in an attempt to avoid femoral artery puncture.

When compared with the IJ and subclavian vein cannulations, femoral catheters are associated with higher risk of infection, thrombosis, and arterial puncture (5). Inadvertent puncture of the femoral artery can be resolved with direct pressure. As with the IJ and subclavian veins, ultrasound has been shown to increase success and reduce complications in femoral catheter placement (11).

ARTERIAL LINES

The preparation for arterial line (A-line) placement begins with sterile preparation of the site, local anesthesia of the insertion site, and positioning of the patient's wrist on a hand board in the dorsiflexed position (14). The radial artery is located 1 to 2 cm from the wrist, between the bony head of the distal radius and the flexor carpi radialis tendon. The two techniques for A-line placement (over-the-wire and over-the-needle) begin with palpation of the artery and location of the radial pulse followed by entry of the needle at a 30°–45° angle. In the over-the-wire technique, the catheter is advanced through the artery until a flash of blood is noted. At this point, both the needle and the catheter are advanced 1 cm deeper in order to completely transect the artery. After this, the needle is removed and the catheter is lowered until it is almost parallel with the skin. The catheter is pulled gently until pulsatile blood is noted. At this point, the guidewire is introduced, and then the catheter is advanced into the artery through the guidewire. Finally, pressure should be placed proximal to the catheter and the wire removed. For the over-the-needle technique, the needle is advanced slowly through the skin until a flash of blood is seen. The needle is then lowered 10°–15°, and the catheter is advanced into the artery. The catheter is then secured in place.

Continuous blood pressure monitoring is mandatory in patients requiring vasopressors.

Use of the radial artery is commonly employed due to the dual arterial supply of the hand by the ulnar artery (7). However, the radial artery is susceptible to vasospasm and hematoma formation after multiple cannulation attempts. In a

Figure 2 Placement of a central venous catheter in the right subclavian vein. Source: With permission from New England Journal of Medicine.

prospective randomized study by Levin et al. comparing ultrasound-guided radial artery cannulation versus the traditional palpation technique, two-dimensional ultrasound-guided catheterization was superior to palpation for first insertion attempt and overall total number of attempts (15).

CENTRAL VENOUS PRESSURE

One of the main goals of hemodynamic monitoring is to predict which patients will improve their hemodynamic conditions when a fluid bolus is given. This is of outmost importance since excessive fluid administration may lead to unwanted consequences such as cerebral edema, pulmonary edema, heart edema with diastolic dysfunction, bowel edema and intra-abdominal hypertension, and poor wound and anastomotic healing, among others. Obstetricians are faced with this challenge frequently when deciding to administrate fluids to a pre-eclamptic patient.

Central venous pressure (CVP) is the pressure measured through a CVC placed with its distal tip at the junction of the superior vena cava and the right atrium. Such a catheter is attached to a fluid-filled system and a pressure transducer, producing a characteristic waveform in the monitor (Fig. 3).

Clinical use of CVP requires careful attention in order to achieve proper measurements (16). The anatomical site to “zero” the line should be 5 cm vertically below the sternal angle, which corresponds to the midpoint of the right atrium.

If one desires to estimate cardiac preload, the CVP measurement should be done at the base of the “c” wave (Fig. 3). This corresponds to the last pressure estimate prior to ventricular contraction, thus yielding the best estimate of cardiac preload. In cases where the “c” wave of the CVP tracing cannot be identified, the base of the “a” wave may be used. Alternatively, this point may be identified by tracing a vertical line from the Q wave of the continuous EKG tracing in the monitor (16). All estimates should be performed at the end of expiration (17). Simply reading the CVP value displaced in the monitor is inaccurate since this is an average of pressures taken over time irrespective of the diastolic cycle.

CVP variations during the respiratory cycle have classically been associated with hypovolemia. In spontaneously breathing patients and in mechanically ventilated patients with triggered efforts, an inspiratory fall in CVP (measured at the base of the “c” or “a” wave) greater than 1 mmHg may indicate fluid responsiveness.

Figure 3 Central venous pressure waveform. Wave “a” corresponds to atrial contraction and wave “c” is secondary to the closure of the tricuspid valve and the isovolumetric contraction of the right ventricle, which displaces the tricuspid leaflets upward, causing an increase in pressure in the right atrium. Wave “v” corresponds to passive right atrium filling during early diastole. The correct place to measure CVP is shown by the asterisks, which approximate the base of wave “c”

A CVP tracing with a prominent “y” descent usually indicates that cardiac output (CO) will not increase with fluid administration (16).

CVP has been used as a monitor of central blood volume and thus preload to the heart. This latter concept has been challenged during the last years as CVP is affected by multiple factors such as right ventricular compliance, tricuspid valve regurgitation, positive pressure ventilation, pericardial disease, vascular tone, and arrhythmias (18).

For example, patients with decreased right-sided CO secondary to a myocardial infarction or pulmonary hypertension or an acute pulmonary embolism will all have elevated CVP, and this will show no correlation with the overall volume status. The same situation is typical in patients with cardiac tamponade, where despite elevated CVP values, initial therapy includes aggressive fluid resuscitation.

The major pitfall of CVP in predicting fluid responsiveness is that it is a static preload measurement. The latter means that a specific value of CVP will not necessarily guarantee an increase in CO after a fluid challenge. A recent trial showed that among patients with a CVP of less than 8 mmHg, the positive predictive value for fluid responsiveness, defined as an increase in cardiac index of $\geq 15\%$, was only 47% (19).

A systematic review of the literature showed that there was no association between CVP values and circulating blood volume and that CVP does not predict fluid responsiveness (20). Furthermore, the receiver operator characteristic curve for CVP was only 0.56. This means that a low CVP will predict an increase in CO in only 56% of the cases. This is similar to flipping a coin!

Thus, a patient is equally likely to respond to fluid with a low or a high CVP (20). Despite lack of scientific evidence, the 2008 Surviving Sepsis Campaign still recommends utilizing CVP as an end point for fluid resuscitation in the setting of septic shock (21).

During early sepsis (first 6 hours of presentation), the use of a CVP-guided protocol of resuscitation as shown by Rivers et al. did improve outcomes (22). We believe that the use of CVP in these first hours of sepsis management is acceptable.

Most of the recent literature concurs in that CVP should not be used to guide fluid management. However, CVP may still be useful as a marker of right ventricular performance in cases of right-sided myocardial infarction, post heart transplant, pulmonary hypertension, and acute pulmonary embolism (20).

PULMONARY ARTERY OCCLUSION PRESSURE

The pulmonary artery catheter (PAC) allows one to measure multiple hemodynamic variables. The catheter is 110 cm long

and contains a variable amount of ports. An infusion port is usually present. The proximal port connects to the proximal lumen, which is located 30 cm proximal to the distal end of the catheter. This port, when connected to a fluid-filled system and a pressure transducer, permits continuous measurement of the CVP. The distal port allows continuous measurements of pulmonary artery pressures. An additional port allows a syringe (with 1.5 mL of air) to be connected to it. By inflating this port, one inflates the balloon present in the distal part of the catheter. When this happens, blood flow after that point will be interrupted due to vascular occlusion secondary to the inflated balloon. The pressure immediately distal to the balloon, when inflated, is the pulmonary artery occlusion pressure (PAOP). PAOP is also measured at the end of expiration. This, theoretically, is a surrogate of the left ventricular filling pressure at the end of diastole since a “column” of blood will be present between the site of balloon inflation (in a branch of the pulmonary arterial system), the pulmonary venous system, the left atrium, the mitral valve, and, finally, the left ventricle (LV).

The catheter also allows measuring the CO by the thermodilution method. Once the CO is calculated, the systemic vascular resistances (SVR) are also calculated as follows: $SVR = (MAP - CVP/CO) \times 80$, where MAP is the mean arterial pressure.

The PAC is advanced through a larger introducer, which is placed as any other central line with the Seldinger technique. The two preferred approaches to float a Swan–Ganz catheter are the right IJ vein and the left subclavian vein. As the catheter is advanced, characteristic waveform changes will occur as its tip enters the right atrium, right ventricle, and pulmonary artery as well as when the inflated balloon occludes the vessel completely (PAOP). Figure 4 depicts these waveform changes.

Complications with the use of the PAC include pneumothorax, hemothorax, arrhythmias, pulmonary infarcts and hemorrhages, and heparin-induced thrombocytopenia, among others. Extreme care is recommended when obtaining a PAOP value in the patient with severe pulmonary hypertension, where pulmonary hemorrhage may occur while obstructing blood flow with the inflated balloon.

Recent literature has questioned the benefit of the PAC in clinical practice. A recent trial involving patients with acute lung injury and adult respiratory distress syndrome (ARDS) showed that the PAC did not improve outcomes as compared with patients managed with a conventional CVP line (23). Another prospective cohort study including patients with severe sepsis also showed no benefit from the PAC (24). A randomized multicenter control study including patients with shock, ARDS, or both similarly showed no benefit from the PAC (25). Similar findings have been reported in the setting of

Figure 4 As the pulmonary artery catheter is advanced, the waveform analysis in the monitor will display typical patterns according to the anatomical site, where the tip is located. When the tip of the pulmonary artery catheter (PAC) is in the superior vena cava and right atrium, the waveform corresponds to the central venous pressure tracing. Entrance to the right ventricle leads to a sudden increase in the systolic pressure (15–30 mmHg) with the same diastolic pressure as on the right atrium (0–8 mmHg). As the PAC is advanced more deeply, the pulmonary artery is entered. The latter leads to an increase in diastolic pressure (6–12 mmHg) with the same previous systolic pressure seen in the right ventricle. Also the entrance to the pulmonary artery may be noticed by the appearance of a notch in the waveform caused by the closure of the pulmonary valve. If one keeps advancing the catheter, one will notice in the monitor the tracing of the pulmonary artery occlusion pressure (PAOP) when the balloon obstructs the flow distal to its position. This pressure should generally be less than the pulmonary artery diastolic pressure and is always measured at the end of expiration.

Figure 5 Frank–Starling curve. When the heart operates in the steep portion of this curve, it will respond by increasing stroke volume in the setting of a fluid bolus.

congestive heart failure, general critical illness, and high-risk perioperative patients (26). In trauma patients, most trials have shown no benefit with the use of the PAC to manage resuscitation. Only one trial, to our knowledge, showed a decrease in mortality in a subset of trauma patients (severely injured with severe shock and elderly patients with moderate shock) (27).

The use of PAOP to predict fluid responsiveness has the same limitations discussed previously for the CVP. It is a static measurement of preload and consequently does not predict fluid responsiveness (28). A PAOP of <12 mmHg predicted fluid responsiveness (defined as an increase in cardiac index of at least 15%) in only 54% of critically ill septic patients (19). This is not surprising since PAOP is not a reliable surrogate of the left ventricular filling pressure; it only reflects the internal pressure of the pulmonary veins relative to atmospheric pressure (18). This measurement is also widely affected by multiple factors. Positive end-expiratory pressure increases PAOP. Of particular concern in obstetrics, pre-eclamptic patients frequently have left ventricular diastolic dysfunction. The latter means that the ventricle will be unable to accommodate significant amounts of fluid. This will cause retrograde accumulation of blood, providing high values in PAOP despite a hypovolemic state.

Significant increases in afterload will diminish left ventricular stroke volume. This will also elevate PAOP despite no changes in total blood volume.

Again, the adequacy of intravascular volume cannot be assessed by one CVP or PAOP (29).

Classic obstetrical literature has attempted to provide a list of clinical scenarios where the PAC is usually indicated (e.g., severe pre-eclampsia not responsive to anti-hypertensive therapy, difficult to manage pulmonary edema, acute kidney injury in the setting of pre-eclampsia); however, it is our opinion that most of these patients can be managed safely without this device.

We favor dynamic measurements of preload responsiveness, where the hemodynamic effect after a fluid challenge can be assessed at the bedside, as will be discussed later in this chapter.

ECHOCARDIOGRAPHY AND FLUID STATUS

The use of transthoracic echocardiography (TTE) may be useful in evaluating hemodynamics. This is an attractive option due to the noninvasiveness of the technique.

Simple subjective assessment of the left ventricular volume by estimating the left ventricular size may aid in guiding fluid therapy (30). A small hyperdynamic LV is typical of hypovolemic patients. Also systolic obliteration of the left ventricular cavity is very suggestive of decreased blood volume (30).

Obviously, certain limitations exist. A large LV end-diastolic area does not always guarantee adequate preload as this may be secondary to LV dilation in the setting of heart failure. On the contrary, a small LV end-diastolic area (frequently suggestive of hypovolemia) may be present in patients with a significant drop in SVR (e.g., sepsis), which favor LV emptying.

Circulating volume status may also be evaluated indirectly by assessing the change in diameter of the inferior vena cava (IVC) during the respiratory cycle (31). During spontaneous breathing, the negative pressure generated in the thoracic cavity during inspiration will favor preload return to the right heart; this will decrease the caliber of the IVC. The more pronounced the respiratory variation in the IVC diameter, the more likely right atrial pressures will be low (and theoretically, the more likely the patient will respond to a fluid bolus). A dilated IVC (diameter more than 2 cm) that does not decrease caliber during spontaneous inspiration is indicative of high right atrial pressures (30).

Figure 6 Example of pulse pressure (PP) variation in an arterial line tracing. In this situation, the double line is the maximal PP, while single line corresponds to the minimal PP. This variation in PP in patients on mechanical ventilation predicts fluid responsiveness.

Figure 7 When oxygen delivery (DO_2) is compromised, tissues will increase extraction of O_2 from hemoglobin. As a consequence, evaluation in the central venous circulation will reveal low hemoglobin oxygen saturation. Abbreviations: CO, cardiac output; Hb, hemoglobin; SVC, superior vena cava.

Some data suggest that using a 12% cutoff in IVC diameter variation with respiration will predict fluid responsiveness with a positive predictive value of 93% (32).

The accuracy of the IVC diameter in predicting fluid responsiveness in patients mechanically ventilated is significantly decreased, as the increased intrathoracic pressures may prevent IVC collapse. Also the application of this method during pregnancy is unknown.

Other methods used to assess LV volume include left ventricular end-diastolic measurements. A left ventricular end-diastolic diameter smaller than 2.5 cm or an LV end-diastolic area less than 55 cm^2 is typically associated with hypovolemia (33).

Just like CVP and PAOP, most of these echocardiographic-based measurements are static measures of volume status and, as such, poorly predict fluid responsiveness (34).

PULSE PRESSURE VARIATION

Since static preload measurements (CVP, PAOP, LV end-diastolic measurements) are no better than random chance at predicting fluid responsiveness, other dynamic approaches have come into clinical practice.

Pulse pressure (PP) is the difference between systolic and diastolic pressures. The use of pulse pressure variation (PPV) to predict fluid responsiveness applies only to patients on mechanical ventilation, not triggering the ventilator, with tidal volumes of 8 to 10 mL/kg, and in sinus heart rhythm.

A recent prospective observational study found that PPV was still an adequate tool to predict fluid responsiveness despite the use of lower tidal volumes (6 mL/kg in setting of ARDS) (34,35).

During conventional mechanical ventilation, each time the ventilator delivers the tidal volume, right atrial pressure will increase. This increase in pressure will decrease preload to the right ventricle in patients who are preload responsive (28). By achieving this, there will be less leftward displacement of the interventricular septum, allowing more filling of the LV. Tidal volume delivery also squeezes the lungs, increasing volume return to the LV through the pulmonary venous system. In addition, during this inspiratory phase, the left ventricular afterload will be significantly decreased. All of the above will lead to an increase in LV stroke volume during inspiration (reversed pulsus paradoxus) (36). This means that during inspiration, PP will increase. If the variation in PP during inspiration and expiration is larger than 13%, patients will likely respond to fluids. Ventilator-induced cyclic changes in preload to both right and left ventricles are more pronounced when the ventricles operate on the steep portion of the Frank-Starling curve (Fig. 5).

In patients with an arterial line in place, the latter is relatively easy to identify at the bedside. The arterial line tracing is evaluated over a period of 30 to 60 seconds, and the maximal and minimal PPs are recorded (Fig. 6). The PPV is then calculated by using the following formula: $PPV = \frac{\text{Max PP} - \text{min PP}}{(\text{max PP} + \text{min PP})0.5} \times 100$. If PPV is $>13\%$, patients will respond to a fluid bolus (28).

The latter concept may also be applied to the pulse oximetry plethysmographic curve (36). A sharp waveform with a clear dicrotic notch indicates adequate peripheral perfusion, while a sinus-shaped waveform reflects poor perfusion. Actually, the height of the pulse oximetry curve correlates well with PP (36). Consequently, in mechanically ventilated patients who meet

the same criteria cited above for PPV evaluation, respiratory-induced changes in the plethysmographic waveform is a useful method to predict fluid responsiveness.

PASSIVE LEG RAISING

Passive leg raising (PLR) is a useful method to predict fluid responsiveness. Unlike PPV, PLR may be used in patients who are spontaneously breathing or triggering the ventilator. It may also be used despite the presence of cardiac arrhythmias (34).

This test simply involves elevation of the lower extremities at a 30° to 45° angle for 1 minute. The maneuver causes a 300-mL blood bolus that will persist for up to 3 minutes before intravascular volume redistribution happens (28). Ideally, while doing PLR, some form of real-time measurement of blood flow should be available (e.g., esophageal Doppler monitoring allowing continuous measurement of both diameter and blood velocity in the aorta) in order to evaluate the immediate cardiovascular response to the maneuver. Patients who will respond to fluids (by increasing CO) will display an increase in aortic blood flow and diameter.

The latter technology is not widely available. A recent trial utilized TTE to evaluate response to PLR. A stroke volume increase higher than 13% after PLR predicted fluid responsiveness with a sensitivity of 77% and a specificity of 100% (37).

During pregnancy, mechanical compression by the uterus to the IVC may alter the response to this maneuver. No data are available about this issue.

SVO₂ AND SCVO₂

SvO₂ is the saturation of hemoglobin measured from a sample of blood obtained from the pulmonary circulation. This may be accomplished by sampling through the distal lumen of a PAC. The normal value is >65%.

ScvO₂ is the hemoglobin saturation of a sample obtained at the junction of the superior vena cava and the right atrium. Collecting this sample only requires the presence of a regular CVC. The normal value is >70%.

During pregnancy, there is an increase in basal metabolic rate of 20% as compared with an increase in CO of 40% to 50%. The higher the CO, the faster the red blood cell travels through the distal tissues. The latter will decrease the available time of perfused tissues to extract oxygen from hemoglobin, leading to higher hemoglobin saturation in the venous circulation. Consequently, one would expect that during pregnancy, SvO₂ (and probably ScvO₂) would be higher than nonpregnant values. However, Hankins and colleagues reported an SvO₂ of 72% in a group of normal pregnant women who received a PAC during the third trimester (38). This is not different from the values reported in healthy nonpregnant individuals.

Decreased values associate with poor oxygen delivery since hypoperfused tissues will extract more oxygen from hemoglobin, leading to low hemoglobin saturations in the venous pool (Fig. 7).

The clinician may attempt to correct low values by increasing resuscitation efforts including fluid resuscitation, blood transfusions, inotropic support, and decreasing

oxygen consumption through mechanical ventilation and sedation.

ScvO₂ monitoring has decreased mortality when used to guide resuscitation during early sepsis management (22).

No data exist on the use of these parameters to guide resuscitation during pregnancy.

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