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Hemodynamic Monitoring in Intensive Care

Abstract: The goal of hemodynamic monitoring is to assess the magnitude of physiological derangements in critically ill patients and to institute measures to correct the imbalance. Basic hemodynamic monitoring consists of clinical examination, invasive arterial monitoring, central venous pressure monitoring, hourly urine output, central venous oxygen saturation and echocardiography. In the presence of positive pressure ventilation with PEEP, central venous and pulmonary artery occlusion pressures may be falsely elevated and need to be interpreted with caution. The fluid challenge is the only way to interpret Central Venous Pressure (CVP) or Pulmonary artery occlusion pressure (PAOP). Dynamic indices of fluid responsiveness such as the pulse pressure variation and stroke volume variation can guide decision making for fluid resuscitation. The hourly urine output is a cheap, simple and indirect method of assessing adequacy of cardiac output and tissue perfusion. Cardiac output is traditionally measured using the pulmonary artery catheter; less invasive methods including the pulse contour analysis and arterial pulse pressure derived methods are now available. It is essential to determine whether hemodynamic therapy is resulting in adequate supply of oxygen to the tissues proportionate to their demand. Mixed and central venous oxygen saturation and lactate levels are commonly used to determine the balance between oxygen supply and demand.

The ultimate purpose of hemodynamic monitoring in anesthesia and intensive care is to assess the delivery of adequate metabolic substrates to various organs and the removal of the end products of metabolism. Under physiological condition the internal milieu is maintained by the organism's own homeostatic mechanisms. The goal of hemodynamic monitoring is to assess the magnitude of these derangements and to institute measures to correct the imbalance.

The clinician uses a variety of surrogates at the bedside to assess the adequacy of supply of substrates in relation to demand. These can range from basic monitoring such as heart rate, non-invasive blood pressure, peripheral temperature, urine output to more advanced and invasive monitoring like the measurement of pulmonary artery pressure, cardiac output and mixed venous oximetry.

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Clinical examination, invasive arterial monitoring, central venous pressure monitoring, hourly urine output, central venous oxygen saturation and echocardiography.

Invasive Arterial Blood Pressure Measurement ¹

Indications: There are two main indications for arterial cannulation. 1. Continuous monitoring of blood pressure. 2. Need for frequent sampling. Some newer methods of continuous cardiac output measurement (PiCCO, LidCO, Arterial Pressure based Cardiac Output (APCO) also use the shape of the arterial pressure waveform for calculation of cardiac output.

Central Venous Pressure Monitoring

Measurement of the Central Venous Pressure (CVP) gives important hemodynamic information regarding the status of the cardiovascular system and response to therapy. Clinically, measurement of the CVP is used for two purposes (1) to gain information about cardiac function, and (2) to gain information about the adequacy of vascular volume. It performs this dual function

because physiologically the right atrial pressure is the interface between the two major determinants of cardiac output: cardiac contractility and venous return. Because CVP influences and is influenced by, both of these physiologic functions, it is an extremely useful measure of cardiovascular function. However, the process of hemodynamic measurements makes several assumptions regarding the cardiovascular system. Hence, the CVP can be confusing and difficult to interpret. The most common reasons for CVP measurement are to help determine the state of volume. A single measurement of the CVP helps somewhat in defining circulatory status but leaves considerable overlap in possible interpretations.

CVP Measurement and Intrathoracic Pressure²

Since the central veins are located inside the thorax, CVP measurements are influenced by changes in intrathoracic pressure. Consequently, the CVP fluctuates with respiration, decreasing with a spontaneous inspiration and increasing with a positive pressure respiration. In order to minimize the effects of respiration, the CVP measurement should be taken at end exhalation when the muscles of respiration are at rest and intrathoracic pressure is stable at its resting level (Fig. 1).

Positive End Expiratory Pressure (PEEP) applied to the airway at the end of exhalation, may be partially transmitted to the intrathoracic structures. Therefore, a CVP measured while patient is receiving PEEP may be higher than under the same cardiovascular circumstances if he was not receiving PEEP. A more accurate measure of right heart filling pressure is the transmural right atrial pressure, that is intravascular right atrial pressure minus intrathoracic pressure. Under most circumstances, measuring CVP referenced to atmosphere is adequate because intrathoracic pressure remains constant. However, when PEEP is applied, pleural pressure increases more than right atrial pressure, so that transmural right atrial pressure (true filling pressure) decreases. This effect is rarely significant if the PEEP is less than 7.5 cm H₂O. The cardiovascular effects of such an increase in CVP are twofold: a decrease in venous return and a decrease in cardiac output. This may be erroneously interpreted as a deterioration in cardiac function. An obvious solution to this problem is to measure the CVP only when the PEEP has been removed. However, the beneficial effect of PEEP on gas exchange is lost very quickly when it is removed and may take a prolonged period to recover when PEEP is reapplied. This subjects the patients to the dangers of hypoxemia. The only accurate method of compensating for the effects of PEEP on central pressure measurements is to measure the transmural pressures. Unfortunately, simple accurate measurement of intrathoracic pressure in the clinical setting is difficult. From a practical standpoint, PEEP does not usually cause a large change in the CVP, especially in patients with stiff lungs. It is best not to remove PEEP for measurements, recognizing that the numbers obtained may be slightly higher than would be found without PEEP.

Hence the only practical way of interpreting the CVP is using a Fluid Challenge.

Fluid Challenge

Rapid changes in vascular volume can help further define the cardiovascular status if the time course of compliance changes is remembered. This is the principle of the "fluid challenge". It consists of 4 components:

1. *Type of fluid:* Usually normal saline
2. *Rate of infusion:* 250-500 ml over 20-30 minutes
3. *Desired therapeutic response:* The parameters are set empirically by the physician. These could be mean arterial pressure (MAP >70 mmHg), HR < 100/min, hourly urine output > 0.5ml/kg/hr.
4. *Danger / safety limits:* Again set empirically by the physician. e.g. CVP 16 mmHg.

Thus after a fluid challenge is given, if neither the therapeutic nor safety limits are reached, the fluid challenge is repeated. If either the target or safety limit is reached, the fluid challenge is terminated.

Pulmonary Artery Occlusion Pressure (PAOP) Monitoring

Swan and Ganz introduced the balloon tipped pulmonary artery catheter into clinical practice in early seventies. This brought the catheter out of the domain of radiologists and at the bedside of the patients in intensive care. Notwithstanding the controversies regarding the utility of pulmonary artery catheter in improving outcome, the clinician in the operating room and ICU needs to be conversant with the use of pulmonary artery catheter.

Physiology of Pulmonary Artery Pressure, Pulmonary Artery Wedge Pressure

The 3 most important measurements obtained from the PAC are:

1. Pulmonary Artery Occlusion Pressure (PAOP)
2. Thermodilution cardiac output
3. Mixed venous oxygen saturation

When a PAC floats to the wedge position, the inflated balloon at its tip isolates the distal pressure monitoring orifice from upstream PAP. Blood flow ceases between the catheter tip and a junction point where pulmonary veins draining the occluded pulmonary vascular region join other veins in which blood still flows towards the left atrium. A continuous static column of blood now connects the occluded PAC tip to this junction point (j point) in the pulmonary veins near the left atrium. Thus, wedging finally extends the catheter tip to measure the pressure at the point where blood flow resumes on the venous end of pulmonary circuit. Thus, PAOP provides an accurate and indirect measurement of both pulmonary venous pressure and Left Atrial Pressure (LAP). For PADP/PAOP to be a valid estimate of left ventricular filling pressure, a static column of blood must exist between the tip of the wedged catheter and the 'j' point. At the microcirculatory levels, this connecting channel consists of thin pulmonary capillaries, which are subject to extramural compressive forces exerted by surrounding alveoli. West has described 3 physiologic lung zones that are based on the gravitationally determined relations between PAP, pulmonary venous pressure and alveolar pressure (Fig. 2). In zones 1 and 2 alveolar pressure exceeds both PAP and pulmonary venous pressure and pulmonary venous pressure respectively. Thus if the catheter is positioned in any of these zones it will monitor alveolar or airway pressure instead of the vascular pressure in the left atrium. Fortunately, in most clinical settings where a PAC is inserted the patient is in supine position which facilitates zone 3 formation.

Criteria for confirming the placement of the PAC are:

1. A tracing consistent with the atrial pressure waveform.
2. A mean wedge pressure lower than mean PA pressure.
3. Arterialized blood aspirated from the catheter tip with the balloon inflated.
4. Free flow when the catheter is wedged (as determined by the absence of "over-wedging" and by ability to aspirate blood through the catheter tip).

Airway Pressure and Pulmonary Artery Occlusion Pressure

Since a continuous column of fluid is required for a stop-flow PAC to sense pulmonary venous pressure at the 'j' point, if alveolar pressure (P_{aw}) increases too much above left atrial pressure, the pulmonary vasculature in the zone of the occluded vessels may collapse such that the wedge pressure senses more airway pressure than the wedge pressure. Such conditions classically occur in West zones 1 and 2. When P_{aw} increases enough relative to left atrial pressure, the distal tip of the PAC senses P_{alv} rather than the wedge pressure. However, such conditions are easy to

identify at the bedside because the respiratory increases in wedge pressure during this condition exceeds the respiratory swings in PADP.

Pleural Pressure and PAOP

Ventilation causes significant swings in pleural pressure. Pulmonary vascular pressure, when measured relative to atmospheric pressure, will reflect these respiratory changes. To minimize this impact variables are conventionally measured at end-expiration. An index of transmission has been described to account for the effects of PEEP (Fig. 3). However the fluid challenge remains the best method of assessing the PAOP during mechanical ventilation with PEEP.

Dynamic Methods to Assess Fluid Responsiveness

The magnitude of respiratory changes in left ventricular stroke volume or its surrogates has been proposed to detect volume responsiveness in mechanically ventilated patients. The rationale for using such indices is based on the assumption that the cyclic changes in right ventricular preload induced by mechanical ventilation should result in greater cyclic changes in RV stroke volume when the right ventricle operates on the steep rather than on the flat portion of the Frank-Starling curve. Large changes in left ventricular stroke volume should occur in case of biventricular preload-dependence; while no change in left ventricular stroke volume should occur if at least one of the two ventricles is preload independent. Because a significant response to fluid in terms of increase in cardiac output should occur only under biventricular preload-dependent conditions it has been logically postulated that the magnitude of cyclic changes of stroke volume would correlate with the degree of response to fluid. In patients receiving controlled ventilation, this hypothesis has been confirmed by taking cyclic changes of arterial pulse pressure of the area under the systolic part of a peripheral artery pressure curve as surrogates of cyclic changes of left ventricular stroke volume.

For accurate prediction, patient should be on controlled ventilation with good patient-ventilator synchronicity.

These dynamic indices include Systolic Pressure Variation, Pulse Pressure Variation and Stroke Volume Variation (Figs 4 and 5).

LIMITATIONS OF DYNAMIC PARAMETERS

- Dynamic indices using heart lung interaction cannot be used in patients with spontaneous breathing activity and / or with arrhythmias.
- Volume responsiveness is a physiological phenomenon related to a normal preload reserve since both ventricles of healthy subject operate on the steep portion of the preload/stroke volume relationship. Therefore, detecting volume responsiveness must not systematically lead to the decision to infuse fluid. Such a decision must be based on the presence of signs of cardiovascular compromise and must be balanced with the potential risk of pulmonary edema formation and /or worsening gas exchange.

Measurement of Cardiac Output Using the Pulmonary Artery Catheter

The cardiac output measurement using the pulmonary artery catheter based on the principle of dye indicator dilution method were first proposed in 1890's by Stewart, and later refined by Hamilton. From the cardiac output reading, other hemodynamic parameters and arterial and mixed blood gas values, other hemodynamic and oxygenation parameters can be calculated (Table 1).

Other Methods of Cardiac Output Measurement

PiCCO: With PiCCO bolus transpulmonary thermodilution cardiac output and pulse contour derived continuous cardiac output measurement are obtained.

Principle of transpulmonary thermodilution: The principle is similar to the thermodilution technique used with PAC. Transpulmonary thermodilution is performed by injecting cold saline through the central venous catheter and the cardiac output (bolus) is calculated after change in temperature is sensed by the thermistor in the femoral artery. Thus the indicator (i.e. the cold saline) traverses through the pulmonary circulation, hence the name transpulmonary thermodilution. This value of cardiac output is then used for calibration for deriving cardiac output by pulse contour method.

Principle of pulse contour analysis: The origin of the pulse contour method for estimation of beat-to-beat stroke volume goes back to the classic Windkessel model described by Otto Frank in 1899. The aortic pressure waveform results from the interaction between stroke volume and the mechanical characteristics of the arterial tree. PiCCO uses a three element “Wind-kessel” model to predict instantaneous aortic flow from peripheral arterial pressure waveform. The stroke volume is then calculated from the systolic part of the arterial pressure waveform. The values attributed to model parameters (resistance, compliance and characteristic impedance) are initially estimated according to the patient’s sex and age, and from the pressure waveform. They are then refined following a calibration of mean cardiac output using an indicator dilution technique the transpulmonary thermodilution. For performing transpulmonary thermodilution, a routine central venous catheter and a large arterial catheter with thermistor (placed usually in the femoral artery) is required.

Advantages: The main advantages over the pulmonary artery catheter is PiCCO is far less invasive, easy to place requiring routine skills of placing a central line and arterial line, which the majority of critical care patients have as a matter of course. This in turn leads to far less risk of complications. Since the indicator traverses both the pulmonary and systemic circulation the cardiac output obtained is from the left ventricle and not the right ventricular as with PAC. The measurements obtained from transpulmonary thermodilution are influenced less by the respiratory cycle than the pulmonary artery catheter. The additional advantages are the values of extravascular lung water, global end-diastolic volume and the stroke volume variation (a dynamic measure of preload).

MEASUREMENT OF INTRATHORACIC BLOOD VOLUME AND EXTRAVASCULAR LUNG WATER

During thermodilution measurement of CO using pulse contour analysis, the following parameters can be obtained:

1. Intrathoracic Blood Volume (ITBV)
Consists of
 - a. GEDV (Global End Diastolic volume) = volume of blood within the heart
 - b. PTV = pulmonary blood volume
2. Extravascular Lung Water (EVLW)
Normally 3-7 ml/kg
>7 ml/kg indicates pulmonary edema

Due to limited expansion of the thorax, the intrathoracic blood volume, intrathoracic gas volume and extravascular lung water volumes interact and change proportionally with each other.

ITBV is a volumetric measurement of cardiac preload (ventricular end-diastolic pressure) and in mechanically ventilated patients, is a sensitive indicator of circulating blood volume.

Disadvantages and contraindications: With PiCCO we need to insert an arterial catheter in a big artery (usually femoral), the existing arterial line cannot be used. It may be contraindicated if arterial access is restricted due to femoral artery grafting or severe burns in areas where the catheter would normally have been placed. The PiCCO may give inaccurate thermodilution measurements in patients with intra-cardiac shunts, aortic aneurysm, aortic stenosis, pneumonectomy, and during extracorporeal circulation. When the central venous catheter is placed in the femoral vein there may be an overestimation of cardiac output by 75 mls/minute.

Arterial Pressure based Cardiac Output (APCO)

Principle: This system calculates the stroke volume from the arterial pressure waveform by measuring the standard deviation of blood pressure. This sampling of blood pressure is done at a frequency of 100 Hz. The relationship between the blood flow in the aorta and pressure wave in the peripheral arteries varies from patient to patient and in the same patient from time to time depending on the clinical condition of the patient. The compliance of the aorta and the cardiovascular system varies according to gender, age, height and weight. Similarly the vascular resistance will vary depending on the clinical condition. The new method (APCO) derives the cardiac output by the following formula:

Cardiac output = Stroke Volume \times HR

CO = $[f(\text{compliance, resistance}) \cdot \sigma_p] \cdot \text{HR}$

CO = $K \times \text{Standard Deviation (BP)}$

i.e. Stroke volume = $[f(\text{compliance, resistance}) \cdot \sigma_p]$

Here, $[f(\text{compliance, resistance})]$ is the constant calculated by the system from arterial compliance and vascular resistance. This constant is recalculated by the system every 10 mins. This value is patient specific and the system calculates it using patient characteristics like age, gender, weight and height using method described by Langewouters.

Advantages: Other systems which use the arterial pressure waveform for calculating continuous cardiac output, need to identify the diastolic notch to find out the systolic part of the arterial waveform. In some situations, for example, in patients with extreme vasodilatation, there might be a peripheral run-off of blood with disappearance of diastolic notch and the monitor cannot calculate the area under the curve. Since APCO takes several samples, it does not need to know the position of the diastolic notch and can still give accurate beat to beat CO. It also means that there is no influence of mean arterial pressure in calculation of cardiac output. Multiple sampling at various pressure points means that the SD (BP) is not going to be affected by random variations in the arterial pressure signal lending stability and reduction in the artifactual noise which can affect the algorithm.

Disadvantages: The main problem with APCO is that it is not validated extensively. Furthermore the performance of the system is likely to be compromised in the presence of following conditions: artifacts in the arterial pressure waveform, compromised arterial catheter, aortic regurgitation, and intense vasoconstriction. We are not yet sure what effects arrhythmias have on the accuracy of APCO. With other pulse contour methods, the accuracy is lost in presence of arrhythmias.

OXYGEN SUPPLY/DEMAND BALANCE

Shock is defined as the disruption of the balance between oxygen demand and supply to the tissues. A low DO_2 can be because of anemia, hypoxia, low cardiac output or maldistribution of blood flow in the microcirculation. VO_2 can be increased in sepsis and systemic inflammation. Oxygen utilization by the tissues can be impaired in sepsis and some forms of poisoning (cytopathic hypoxia). We thus need to assess the balance between oxygen supply and demand.

Mixed Venous and Central Venous Oxygen Saturation

$$SvO_2 = SaO_2 - VO_2 / (CO \times Hb \times 1.34)$$

SvO_2 estimates all components of DO_2 . It reflects CO if VO_2 and Hb are constant, and most importantly, it reflects the balance between oxygen supply and demand. An SvO_2 below 65% implies low oxygen delivery, while a value below 60% indicates that there is a serious risk of tissue hypoxia if corrective measures are not taken. A low SvO_2 (< 40 %) implies critical oxygen supply / demand imbalance. In some disease states, cells in some tissues are unable to assimilate and/or process the needed oxygen. If SvO_2 is high(>80%) then the demand has declined, the O_2 supply has increased, or the cells are unable to utilize oxygen.

Thus a falling or low SvO_2 is an important indicator that the oxygen delivery is compromised and is deficient relative to the needs of the tissues.

In critically ill patients with shock, global tissue hypoxia is a key development preceding multiorgan failure and death. When sepsis progresses to severe sepsis and shock, circulatory abnormalities lead to an imbalance between systemic oxygen delivery and oxygen demand, resulting in global tissue hypoxia. The transition to serious illness occurs during the critical period when definitive recognition and treatment provide maximum benefit in terms of outcome.

Early hemodynamic assessment on the basis of physical findings, vital signs, CVP and urine output in sepsis fails to detect this persistent occult tissue hypoxia. Evidence suggests that 50% of critically ill patients presenting in shock who were resuscitated to normal vital signs continued to have increased lactate and abnormally low SvO_2 , indicating anaerobic metabolism and oxygen debt.

Measurement of mixed venous oxygen saturation (SvO_2) from the pulmonary artery is thus an indirect index of tissue oxygenation. In myocardial infarction, decreased SvO_2 has been found to be indicative of current or imminent cardiac failure. Decreased SvO_2 values predict poor prognosis after cardiovascular surgery, in severe cardiopulmonary disease, and in septic or cardiogenic shock. This led to development of the fiberoptic Pulmonary Artery (PA) catheter for continuous measurement of SvO_2 by reflection spectrophotometry.

However sampling of mixed venous blood requires insertion of a Pulmonary Artery catheter, which is an invasive procedure with risks, and is not universally used. An alternative is to measure central venous oxygen saturation ($ScvO_2$). Central venous catheterization is a simpler and safer procedure, and is commonly used. In this case a catheter is positioned in the superior vena cava or upper right atrium. The $ScvO_2$ is generally greater than SvO_2 with a difference of 5-18%. However, the use of $ScvO_2$ was not considered appropriate, especially in shock. The blood from the hepatosplanchnic circulation in shock is considerably desaturated, when gut blood flow is reduced and with increased oxygen extraction by the gut. This is not reflected in the superior vena cava blood, which may show higher $ScvO_2$.

However for clinical decision making, the exact value of $ScvO_2$ is not important. Whenever the SvO_2 is critically low, $ScvO_2$ is critically low as well and the $ScvO_2$ closely parallels the trend of SvO_2 in critically ill patients. It can thus be used as a convenient surrogate for the SvO_2 . Under shock conditions, a low $ScvO_2$ is consistently associated with even lower SvO_2 . Investigators have shown that $ScvO_2 < 60\%$ was associated with heart failure, shock or a combination of the two.

The $ScvO_2$ is an important clinical parameter and can be considered a reliable indicator of a life threatening oxygen imbalance. This is extremely important because unlike PA monitoring for SvO_2 , central venous access can be easily obtained in both ICU and non-ICU settings. Central venous catheters with fiberoptic sensors for continuous $ScvO_2$ are now available (PreSep, Edwards). A $ScvO_2 < 70\%$ indicates presence of tissue hypoxia, and therapy directed at

maintaining a ScvO₂ > 70% may reduce the incidence of multiorgan failure and death due to occult tissue hypoxia.

Lactate Levels

Lactate levels often reflect anerobic metabolism due to tissue dysoxia. High and rising levels (> 2mmol/L) are adverse prognostic factors, while falling lactate levels indicate an adequate response to resuscitation of the shocked patient. However lactate levels may increase either due to increased production due to global or local tissue hypoxia, and also due to stimulation of glycolysis and metabolic pathways that accelerate lactate formation in sepsis. High lactate levels may also represent decreased clearance due to reduced liver blood flow or hepatic dysfunction. Thus interpretation of lactate levels may be complicated. Also, arterial lactate levels are a global measure, and regional hypoperfusion of some vascular beds may exist even in the presence of normal lactate levels.

SUGGESTED READING

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MULTIPLE CHOICE QUESTIONS

1. **Shock is best defined as:**
 - A. Hypotension with tachycardia
 - B. Hypotension with tachycardia with cold extremities and low urine output
 - C. Low cardiac output and oxygen delivery
 - D. Oxygen supply inadequate for demand of tissues
 2. **CVP measurement should be made at:**
 - A. End-inspiration
 - B. End-expiration
 - C. At the beginning of inspiration
 - D. Mid-inspiration
 - E. Mid-expiration
 3. **To remove the effect of PEEP on CVP:**
 - A. PEEP value should be subtracted from CVP
 - B. The patient should be disconnected from the ventilator at frequent intervals
 - C. PEEP value should be added to CVP
 - D. Serial trends of CVP should be used instead of single values
 4. **Dynamic parameters of fluid responsiveness do not include:**
 - A. Systolic pressure variation
 - B. Stroke volume variation
 - C. Heart rate variability
 - D. Pulse pressure variation
 5. **For accurate prediction using dynamic parameters of fluid responsiveness, patient should be:**
 - A. Mechanically ventilated, breathing spontaneously with low levels of PEEP
 - B. Mechanically ventilated, breathing spontaneously with high levels of PEEP
 - C. Mechanically ventilated, not breathing spontaneously
 - D. Spontaneously breathing without a ventilator
 6. **The following are good indicators of the balance between oxygen supply and demand, except:**
 - A. Arterial lactate
 - B. Cardiac output
 - C. ScvO₂
 - D. Oxygen consumption measurement
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1. D 2. B 3. D 4. C 5. C 6. D