

Practical Issues of Hemodynamic Monitoring at the Bedside

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The hemodynamic monitoring of a surgical patient acquires a major relevance in high-risk patients and those suffering from surgical diseases associated with hemodynamic instability, such as hemorrhagic or septic shock; however, all surgical patients require the monitoring and evaluation, and sometimes benefit from optimizing their hemodynamic status. Therefore, all surgeons should have a basic understanding of the principles, indications, and therapeutic implications of hemodynamic monitoring.

Rationale for hemodynamic monitoring

The arguments to defend the use of specific types of monitoring techniques can be roughly grouped into three levels based on their level of validation [1]. At the basic level, the specific monitoring technique can be defended based on historical controls. At this level, prior experience using similar monitoring was traditionally used and presumed to be beneficial. The mechanism by which the benefit is achieved need not be understood. The second level of defense comes through an understanding of the pathophysiology of the process being treated. This physiological argument can be stated as “knowledge of how a disease process creates its effect and preventing the process from altering measured bodily functions should prevent the disease process from progressing or injuring remote physiological functions.” Most of the rationale for

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hemodynamic monitoring resides at this level. It is not clear from recent clinical studies in critically ill patients that this argument is valid, primarily because knowledge of the actual processes involved in the expression of disease and tissue injury is often inadequate. The third level of defense comes from documentation that the monitoring device, by altering therapy in otherwise unexpected ways, improves outcome in terms of survival and quality of life. In reality, few therapies done in medicine can claim benefit at this level. Thus, we are left with the physiological rationale as the primary defense of monitoring of critically ill patients.

The physiologic basis for hemodynamic monitoring

On a philosophical level, one may consider the monitoring of critically ill patients as serving a dual function. First, it can be used to document hemodynamic stability and the lack of need for acute interventions, and second, it can be used to monitor when measured variables vary from their defined baseline values. Accordingly, knowing the limits to which such monitoring reflects actual physiological values is an essential aspect of its utility.

On the physical side, hemodynamic monitoring can be invasive or noninvasive, and continuous or intermittent. Monitoring devices can measure physiologic variables directly, or derive these variables through signal processing. Signal processing does not minimize the usefulness of physiologic variable analysis; it just separates the output data from the patient by the use of the data processor. The most common signal processing physiologic variable measured clinically is the electrocardiogram.

Variables that can be measured noninvasively include body temperature, heart rate, systolic and diastolic arterial blood pressure, and respiratory frequency. Processed noninvasive variables include the electrocardiogram, transcutaneous pulse oximetry (SpO₂), expired CO₂, trans-thoracic echocardiography, and noninvasive respiratory plethysmography. Invasive monitoring reflects intravascular catheter insertion, transesophageal echocardiographic probe insertion, and blood component analysis. Invasive hemodynamic monitoring of vascular pressures is usually performed by the percutaneous insertion of a catheter into a vascular space and transducing the pressure sensed at the distal end. This allows for the continual display and monitoring of these complex pressure waveforms. Similar intrapulmonary vascular catheters can be used to derive thermal signals and mixed venous oxygen (O₂) saturation (SvO₂), needed to assess cardiac output and the adequacy of O₂ delivery, respectively. How useful this hemodynamic information is to diagnosis treatment and prognosis is a function of its reliability, established treatment protocols, and guidelines, and the expertise of the operator. **Box 1** summarizes all the possible unitary and calculated measures derived from invasive hemodynamic monitoring.

Box 1. Physiological variables derived from invasive monitoring and their physiological relevance

Unitary measures

Arterial pressure

- Mean arterial pressure (MAP)
Organ perfusion inflow pressure
- Arterial pulse pressure and its variation during ventilation
Left ventricular stroke volume changes and pulsus paradoxus
Preload-responsiveness (if assessed during intermittent positive pressure ventilation [IPPV])
- Arterial pressure waveform
Aortic valvulopathy, input impedance, and arterial resistance
Used to calculate stroke volume by pulse contour technique

Central venous pressure (CVP)

- Mean CVP
If elevated, then effective circulating blood volume is not reduced
- CVP variations during ventilation
Tricuspid insufficiency, tamponade physiology
Preload-responsiveness (if assessed during spontaneous breathing)

Pulmonary arterial pressure (Ppa)

- Mean Ppa
Pulmonary inflow pressure
- Systolic pulmonary artery pressure
Right ventricular pressure load
- Diastolic pulmonary artery pressure and pulse pressure, and their variations during ventilation
Right ventricular stroke volume, pulmonary vascular resistance
Diastolic pressure tract changes in intrathoracic pressure during ventilation

Pulmonary artery occlusion pressure (Ppao)

- Mean Ppao
Left atrial and left ventricular intraluminal pressure, and by inference, left ventricular preload
Back pressure to pulmonary blood flow
- Ppao waveform and its variation during occlusion and ventilation

Mitral valvulopathy, atrial or ventricular etiology of arrhythmia, accuracy of mean Ppao to measure intraluminal left ventricle (LV) pressure, and pulmonary capillary pressure (Ppc)

Calculated measures

Calculated measures using multiple measured variables including cardiac output by thermodilution (COtd), arterial blood gases (ABG) and mixed venous blood gases (VBG)

Vascular resistances

- Total peripheral resistance = $MAP/COtd$
- Systemic vascular resistance = $(MAP - CVP)/COtd$
- Pulmonary arterial resistance = $(\text{mean Ppa} - Ppc)/COtd$
- Pulmonary venous resistance = $(Ppc - Ppao)/COtd$
- Pulmonary vascular resistance = $(\text{mean Ppa} - Ppao)/COtd$

Vascular pump function

- Left ventricular stroke volume (SVlv) = $COtd/HR$
- Left ventricular stroke work (SWlv) = $(MAP - Ppao)/SVlv$
- Preload—recruitable stroke work = $SWlv/Ppao$

Oxygen transport and metabolism

- Global oxygen transport or delivery (DO_2) = $CaO_2/COtd$
 - Global oxygen uptake (VO_2) = $(CaO_2 - CvO_2)/COtd$
 - Venous admixture
 - Ratio of dead space to total tidal volume (Vd/Vt) = $Paco_2 / (Paco_2 - Petco_2)$
- Right ventricular (RV) function using RV ejection fraction (EFrv) catheter-derived data
- Right ventricular end-diastolic volume (EDVrv) = $SV/EFrv$
 - Right ventricular end-systolic volume (ESVrv) = $EDVrv - SV$

Abbreviations: HR, heart rate; CaO_2 , arterial O_2 content; CvO_2 , mixed venous O_2 content; $Petco_2$, end tidal CO_2 ; SV, stroke volume; $SV/EFrv$, stroke volume/ejection fraction of right ventricle.

Adapted from Bellomo R, Pinsky MR. Invasive monitoring. In: Tinker J, Browne D, Sibbald W, editors. Critical care—standards, audit and ethics. 26. London: Arnold Publishing Co.; 1996. p. 82–104; with permission.

Arterial pressure monitoring

After pulse rate, arterial pressure is the most common hemodynamic variable monitored and recorded. Blood pressure is usually measured noninvasively using a sphygmomanometer and the auscultation technique. Importantly, very large and obese subjects in whom the upper arm circumference exceeds the width limitations of a normal blood pressure cuff will record

pressures that are higher than they actually are. In such patients, using the large thigh blood pressure cuff usually resolves this problem. Blood pressure can be measured automatically using computer-driven devices (eg, Dynamat) that greatly reduce nursing time. Sphygmomanometer-derived blood pressure measures display slightly higher systolic and lower diastolic pressures than simultaneously measured indwelling arterial catheters, but the mean arterial pressure is usually similar, and the actual systolic and diastolic pressure differences are often small except in the setting of increased peripheral vasomotor tone. If perfusion pressure of the finger is similar to arterial pressure, then both blood pressure and the pressure profile may be recorded noninvasively and continuously using the optical finger probe (Finapres Medical Systems BV, Amsterdam, The Netherlands). Finger perfusion is often compromised during hypovolemic shock and hypothermia, however, limiting this monitoring technique to relatively well-perfused patients.

Accurate and continuous measures of arterial pressure can be done through arterial catheterization of easily accessible arterial sites in the arm (axillary, brachial, or radial arterial) or groin (femoral arterial). Usually neither axillary nor brachial arterial sites are used because of fears of causing downstream ischemia, although there are no data supporting these fears. Arterial catheterization displaying continuous arterial pressure waveforms lends itself to arterial waveform analysis, essential in calculating pulse pressure, pulse pressure variations, and cardiac output.

Why measure arterial pressure?

Arterial pressure is the input pressure for organ perfusion. Organ perfusion is usually dependent on organ metabolic demand and perfusion pressure. With increasing tissue metabolism, organ blood flow proportionally increases by selective local vasodilation of the small resistance arterioles. If cardiac output cannot increase as well, as is the case with heart failure, then blood pressure decreases, limiting the ability of local vasomotor control to regulate organ blood flow. If local metabolic demand remains constant, however, changes in arterial pressure are usually matched by changes in arterial tone so as to maintain organ blood flow relatively constant. This local vasomotor control mechanism is referred to as autoregulation. Cerebral blood flow over the normal autoregulatory range of 65 to 120 mm Hg is remarkably constant. Although autoregulation occurs in many organs, such as the brain, liver, skeletal muscle, and skin, it is not a universal phenomenon. For example, coronary flow increases with increasing arterial pressure because the myocardial O_2 demand increases as the heart ejects into a higher arterial pressure circuit. Furthermore, renal blood flow increases in a pressure-dependent fashion over its entire pressure for similar reasons. As renal flow increases, so does renal filtrate flow into the tubules, increasing renal metabolic demand. Thus, a normal blood pressure does not mean that all organs have an adequate amount of perfusion, because increases in local

vasomotor tone and mechanical vascular obstruction can still induce asymmetrical vascular ischemia.

Determinants of arterial pressure

Arterial pressure is a function of both vasomotor tone and cardiac output. The local vasomotor tone also determines blood flow distribution, which itself is usually determined by local metabolic demands. For a constant vasomotor tone, vascular resistance can be described by the relation between changes in both arterial pressure and cardiac output. The body defends organ perfusion pressure above all else in its autonomic hierarchy through alterations in α -adrenergic tone, mediated through baroreceptors located in the carotid sinus and aortic arch. This supremacy of arterial pressure in the adaptive response to circulatory shock exists because both coronary and cerebral blood flows are dependent only on perfusion pressure. The cerebral vasculature has no α -adrenergic receptors; the coronary circulation has only a few. Accordingly, hypotension always reflects cardiovascular embarrassment, but normotension does not exclude it. Hypotension decreases organ blood flow and stimulates a strong sympathetic response that induces a combined α -adrenergic (increased vasomotor tone) and β -adrenergic (increased heart rate and cardiac contractility) effect, and causes a massive adrenocorticotrophic hormone (ACTH)-induced cortisol release from the adrenal glands. Thus, to understand the determinants of arterial pressure one must also know the level of vasomotor tone.

In the intensive care unit setting, arterial tone can be estimated at the bedside by calculating systemic vascular resistance. Using Ohm's Law, resistance equals the ratio of the pressure to flow, usually calculated as the ratio of the pressure gradient between aorta and central venous pressure (CVP) to cardiac output. Arterial tone can also be calculated as total peripheral resistance, which is the ratio of mean arterial pressure to cardiac output. Regrettably, neither systemic vascular resistance nor total peripheral resistance faithfully describes arterial resistance. Arterial resistance is the slope of the arterial pressure-flow relation. The calculation of systemic vascular resistance using CVP as the backpressure to flow has no physiological rationale, and the use of systemic vascular resistance for clinical decision-making should be abolished. Regrettably, both systemic vascular resistance and total peripheral resistance are still commonly used in hemodynamic monitoring because they allow for the simultaneous assessment of both pressure and flow, whereas the actual measure of arterial tone is more difficult to estimate.

The determinants of arterial pressure can simplistically be defined as systemic arterial tone and blood flow. Because blood flow distribution will vary amongst organs relative to their local vasomotor tone, and arterial pressure is similar for most organs, measurement of peripheral resistance, by any means or formula, reflects the lump parameter of all the vascular beds, and thus describes no specific vascular bed completely. If no hemodynamic

instability alters normal regulatory mechanisms, then local blood flow will also be proportional to local metabolic demand. Within this construct, the only reason cardiac output becomes important is to sustain an adequate and changing blood flow to match changes in vasomotor tone, such that arterial input pressure remains constant. Because cardiac output is proportional to metabolic demand, there is no level of cardiac output that reflects normal values in the unstable and metabolically active patient; however, as blood pressures decreases below 60 mm Hg mean or cardiac indices decrease below 2.0 l/min/m^2 , organ perfusion usually becomes compromised, and if sustained, will lead to organ failure and death. Presently, only one clinical trial has examined the effect of increasing mean arterial pressure on tissue blood flow [2]. When patients with circulatory shock were resuscitated with volume and vasopressors to a mean arterial pressure range of 60 to 70, 70 to 80, or 80 to 90 mm Hg, no increased organ blood flow could be identified above a mean arterial pressure of 65 mm Hg. Clearly, subjects who have prior hypertension will have their optimal perfusion pressure range increased over normotensive patients. Thus, there are no firm data supporting any one limit of arterial pressure or cardiac output values or therapeutic approaches based on these values as more beneficial than any other. Accordingly, empiricism is the rule regarding target values of both mean arterial pressure and cardiac output. At present, the literature suggests that maintaining a previously nonhypertensive patient's mean arterial pressure greater than 65 mm Hg by the use of fluid resuscitation and subsequent vasopressor therapy is an acceptable target. Previously hypertensive subjects will need a higher mean arterial pressure to insure the same degree of blood flow [2]. There is no proven value in forcing either arterial tone or cardiac output to higher levels to achieve a mean arterial pressure above this threshold. In fact, data suggest that further resuscitative efforts using vasoactive agents markedly increase mortality [3], and the relatively new concept of "delayed" and "hypotensive resuscitation" for traumatic hemorrhagic shock has shown improved outcome in some clinical and experimental studies [4–6]. Those studies, however, were in trauma patients who had penetrating wounds and no immediate access to surgical repair. Once a patient is in the hospital and the sites of active bleeding addressed, then aggressive fluid and pressor resuscitation is indicated.

Arterial pressure variations during ventilation

The majority of the critically ill surgical patients treated in the ICU are usually on mechanical ventilation. Ventilation-induced arterial pressure variations have been described since antiquity as *pulsus paradoxus*. Inspiratory decreases in arterial pressure were used to monitor both the severity of bronchospasm in asthmatics and their inspiratory efforts [7].

Recently renewed interest in the hemodynamic significance of heart-lung interactions has emerged. The commonly observed variations in arterial

pressure and aortic flow seen during positive-pressure ventilation have been analyzed as a measure of preload responsiveness [8]. The rationale for this approach is that positive-pressure ventilation-induced changes in either systolic arterial pressure (used to describe pulsus paradoxus), arterial pulse pressure, or stroke volume can predict in which subjects cardiac output will increase in response to fluid resuscitation. Ventilation-induced changes in systolic arterial pressure (pulsus paradoxus) and arterial pulse pressure are easy to measure from arterial pressure recordings. The greater the degree of systolic arterial pressure or pulse pressure variation over the respiratory cycle, the greater the increase in cardiac output in response to a defined fluid challenge. Recently, measuring the mean change in aortic blood flow during passive leg raising in spontaneous breathing patients has also proven accurate in predicting preload responsiveness [9].

Although arterial pressure variations are a measure of preload-responsiveness [10], the “traditional” preload measures, such as right atrial pressure (Pra), Ppao, RV end-diastolic volume, and intrathoracic blood volume, poorly reflect preload-responsiveness [11]. In essence, preload is not preload-responsiveness.

Indications for arterial catheterization

The arterial catheter is frequently inserted as a “routine” at the admission of patients to the ICU for continuous monitoring of blood pressure and repetitive measurements of blood gases. There is no evidence to support this exaggerated clinical practice. Although probably the only proven indication for arterial catheterization is to synchronize the intra-aortic balloon of counterpulsation, there are some others indications whereby the information obtained is valuable in the assessment and treatment of the patient, such as cardiovascular instability or the use of vasopressors or vasodilators during resuscitation. The probable indications for arterial catheterization are summarized in **Box 2**. Although arterial catheterization is an invasive procedure that is not free of complications, a recent systematic review of a large number of cases [12] showed that most of the complications were minor, including temporary vascular occlusion (19.7%) and hematoma (14.4%). Permanent ischemic damage, sepsis, and pseudoaneurysm formation occurred in fewer than 1% of cases [12].

Central venous pressure monitoring

Methods of measuring central venous pressure

CVP is the pressure in the large central veins proximal to the right atrium relative to atmosphere. In the ICUs, the CVP is usually measured using a fluid-filled catheter (central venous line or Swan-Ganz catheter) with the distal tip located in the superior vena cava connected to a manometer, or more often to

Box 2. Arterial catheterization

Indications for arterial catheterization

- As a guide to synchronization of intra-aortic balloon counter pulsation

Probable indications for arterial catheterization

- Guide to management of potent vasodilator drug infusions to prevent systemic hypotension
- Guide to management of potent vasopressor drug infusions to maintain a target MAP
- As a port for the rapid and repetitive sampling of arterial blood inpatients in whom multiple arterial blood samples are indicated
- As a monitor of cardiovascular deterioration in patients at risk for cardiovascular instability

Useful applications of arterial pressure monitoring in the diagnosis of cardiovascular insufficiency

- Differentiating cardiac tamponade (pulsus paradoxus) from respiration-induced swings in systolic arterial pressure—tamponade reduces the pulse pressure but keeps diastolic pressure constant. Respiration reduces systolic and diastolic pressure equally, such that pulse pressure is constant.
- Differentiating hypovolemia from cardiac dysfunction as the cause of hemodynamic instability. Systolic arterial pressure decreases more following a positive pressure breath as compared to an apneic baseline during hypovolemia. Systolic arterial pressure increases more during positive pressure inspiration when LV contractility is reduced.

Adapted from Bellomo R, Pinsky MR. Invasive monitoring. In: Tinker J, Browne D, Sibbald W, editors. *Critical Care—Standards, Audit and Ethics*. 26. London: Arnold Publishing Co., 1996. p. 82–104; with permission.

a pressure transducer of a monitor, displaying the waveform in a continuous fashion. CVP can also be measured noninvasively as jugular venous pressure, the height of the column of blood distending the internal and external jugular veins when the subject is sitting in a semireclined position; small elevations in CVP will be reflected by persistent jugular venous distention.

Determinants of central venous pressure

Starling demonstrated the relationship between cardiac output, venous return, and CVP, showing that increasing the venous return (and preload)

increases the stroke volume (and cardiac output [CO]) until a plateau is reached. Although the CVP is clearly influenced by the volume of blood in the central compartment and its venous compliance; there are several physiological and anatomical factors that can influence its measurement and waveform, such as the vascular tone, right ventricular function, intrathoracic pressure changes, tricuspid valve disease, arrhythmias, and both myocardial and pericardial disease. These are summarized in [Box 3](#).

Box 3. Factors affecting the measured CVP

Central venous blood volume

- Venous return/cardiac output
- Total blood volume
- Regional vascular tone

Compliance of central compartment

- Vascular tone
- RV compliance
- Myocardial disease
- Pericardial disease
- Tamponade

Tricuspid valve disease

- Stenosis
- Regurgitation

Cardiac rhythm

- Junctional rhythm
- Atrial fibrillation (AF)
- Atrio ventricular (A-V) dissociation

Reference level of transducer

- Positioning of patient

Intrathoracic pressure

- Respiration
- IPPV
- Positive end-expiratory pressure (PEEP)
- Tension pneumothorax

From Smith T, Grounds RM, Rhodes A. Central venous pressure: uses and limitations. In: Pinsky MR, Payen D. Functional hemodynamic monitoring. Berlin, Heidelberg (Germany): Springer-Verlag; 2006. p. 101; with kind permission of Springer Science and Business Media.

Monitoring central venous pressure

CVP has been used as a monitor of central venous blood volume and an estimate of the right atrial pressure for many years, being wrongly used as a parameter and sometimes goal for replacement of intravascular volume in shock patients. The validity of this measure as an index of RV preload is nonexistent across numerous studies. It has been shown that CVP has a poor correlation with cardiac index, stroke volume, left ventricular end-diastolic volume, and right ventricular end-diastolic volume [13–15].

Although a very high CVP demands a certain level of total circulating blood volume, one may have a CVP of 20 mm Hg and still have an under-filled left ventricle that is fluid responsive. For example, in the setting of acute RV infarction, CVP can be markedly elevated, whereas cardiac output often increases further with volume loading. In reported series, some patients who had low CVP failed to respond to fluids and some patients who had high CVP responded to challenge of fluids [16]. Based on this and the poor correlations described above, it is impossible to define ideal values of CVP; however, there is some evidence that volume loading in patients who have CVP greater than 12 mmHg is very unlikely to increase cardiac output [17]. Thus, the only usefulness of CVP is to define relative hypervolemia, because an elevated CVP only occurs in disease. Two clinical studies [18,19] showed a potential benefit in specific groups of surgical patients (hip replacement and renal transplant patients) in whom CVP was used to guide therapy; however, there is no clinical evidence that CVP monitoring improves outcome in critically ill patients, and attempts to normalize CVP in early goal directed therapy during resuscitation do not display any benefit [20].

Pulmonary artery catheterization and its associated monitored variables

Pulmonary arterial catheterization allows the measurement of many clinically relevant hemodynamic variables (see **Box 1**). One can measure the intrapulmonary vascular pressures including CVP, Ppa, and by intermittent balloon occlusion of the pulmonary artery, Ppao and pulmonary capillary pressure (Ppc). Furthermore, by using the thermodilution technique and the Stewart-Hamilton equation, one can estimate cardiac output and EFrv, global cardiac volume, and intrathoracic blood volume. Finally, one can measure mixed SvO₂ either intermittently by direct sampling of blood from the distal pulmonary arterial port or continuously via fiber optic reflectometry. Assuming one knows the hemoglobin concentration and can tract arterial O₂ saturation (SaO₂), easily estimated by noninvasively by pulse oximetry as SpO₂, one can calculate numerous derived variables that describe well the global cardiovascular state of the patient. These derived variables include DO₂, VO₂, venous admixture (as an estimate of intrapulmonary shunt), pulmonary and systemic vascular resistance, EDVrv and ESVrv, and both RV and LV stroke work index.

Pulmonary artery pressure

The determinants of Ppa are the volume of blood ejected into the pulmonary artery during systole, the resistance of the pulmonary vascular bed, and the downstream left atrial (LA) pressure. The pulmonary vascular bed is a low-resistance circuit with a large reserve that allows increases of cardiac output with minor changes in the Ppa. On the other hand, increases in the downstream venous pressure (eg, left ventricular failure) or in the flow resistance (eg, lung diseases) raise the Ppa. Although increases in cardiac output alone do not cause pulmonary hypertension, having an increased vascular resistance can lead to elevations in Ppa, with changes in cardiac output. Based on these considerations, the Ppa should not be used as a reliable parameter of ventricular filling under several lung diseases that cause changes in the vascular tone and cardiac output. The normal range of values for Ppa are: systolic 15 to 30 mm Hg, diastolic 4 to 12 mm Hg, and mean 9 to 18 mm Hg [21].

Pulmonary artery occlusion pressure

Methods of measuring pulmonary artery occlusion pressure

Numerous studies by physicians have demonstrated that the ability to accurately measure Ppao from a strip chart recording or a freeze-frame snapshot of the monitor screen is poor. Many initiatives have been put into place to educate physicians and nurses, but the reality is that because the pressure measured also reports changes in intrathoracic pressure, a value which is always changing, the accuracy of Ppao measures is likely to remain poor.

The Ppao value is thought to reflect the LV filling because of the unique characteristic of the pulmonary circulation. Balloon inflation of the pulmonary artery catheter (PAC) forces the tip to migrate distally into smaller vessels until the tip occludes a medium-sized (1.2 cm diameter) pulmonary artery. This occlusion stops all blood flow in that vascular tree distal to the occlusion site, until such time as other venous branches reconnect downstream to this venous draining bed. The point where such parallel pulmonary vascular beds anastomose is about 1.5 cm from the left atrium. Thus, if a continuous column of blood is present from the catheter tip to the left heart, then Ppao measures pulmonary venous pressure at this first junction, or J-1 point, of the pulmonary veins [22]. As downstream pulmonary blood flow ceases, distal pulmonary arterial pressure falls in a double exponential fashion to a minimal value, reflecting the pressure downstream in the pulmonary vasculature from the point of occlusion. The Ppa value where the first exponential pressure decay is overtaken by the second longer exponential pressure decay reflects Ppc measures, useful in calculating pulmonary arterial and venous resistances. Importantly, the column of water at the end of the catheter is now extended to include the pulmonary vascular

circuit up to this J-1 point of blood flow. Because the vasculature is compliant relative to the catheter, vascular pressure signals dampen relative to the nonoccluded Ppa signal. Thus, the two primary aspects of Ppao measures that are used to identify an occluded pressure are the decrease in diastolic pressure values to less than diastolic Ppa and the damping of the pressure signal (Fig. 1). If one needed further validation that the catheter is actually in an occluded vascular bed, then one could measure the pH, pCO₂, and pO₂ of blood sampled from the occluded distal tip of the catheter. Because the sampled blood will be from the stagnant pool of blood, its removal will make it be pulled back into the PAC from the pulmonary veins. Because the blood sampled will have crossed the alveolar capillaries twice, its pCO₂ will be lower than arterial pCO₂, and its pO₂ higher, as a result of the law of mass action.

Pleural pressure and Ppao

Although one may measure Ppao accurately relative to atmosphere, the heart and large vessel pulmonary vasculature live in an intrathoracic compartment and sense pleural pressure (Ppl) as their surrounding pressure. Ventilation causes significant swings in Ppl. Pulmonary vascular pressures, when measured relative to atmospheric pressure, will reflect these respiratory changes in Ppl. To minimize this “respiratory artifact” on intrathoracic vascular pressure recordings, measures are usually made at end-expiration. During quiet spontaneous breathing, end-expiration occurs at the highest vascular pressure values, whereas during passive positive-pressure breathing, end-expiration occurs at the lowest vascular pressure values. With assisted ventilation or with forced spontaneous ventilation, it is often difficult to define end-expiration [23]. These limitations are the primary reasons for inaccuracies in estimating Ppao at the bedside.

Even if measures of Ppao are made at end-expiration and Ppao values reflect a continuous column of fluid from the catheter tip to the J-1 point, these Ppao measures may still overestimate Ppao if Ppl is elevated at end-expiration. Hyperinflation caused by air trapping, dynamic hyperinflation, or the use of extrinsic PEEP will all increase end-expiratory Ppl to a varying degree as a function of airway resistance and lung and chest wall

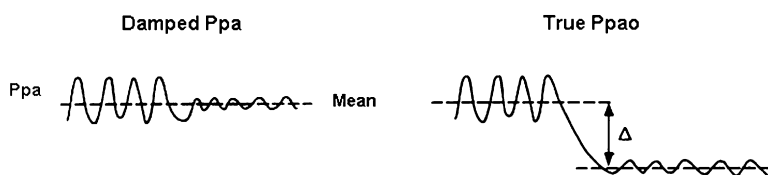


Fig. 1. This figure represents the differential characteristics of a damped Ppa and a true wedge or Ppao during the balloon inflation. Notice the flattening of the waveform on the damped Ppa tracing (*left*) without a decrease of the mean pressure, whereas in the true Ppao (*right*) there is a substantial decrease in the mean pressure.

compliance. It is not possible to predict with accuracy the degree to which increases in PEEP will increase Ppl. Because differences in lung and chest wall compliance exist among patients and within the same patient over time, one cannot assume a fixed relation between increases in airway pressure (Paw) and Ppl [24].

Why measure pulmonary artery occlusion pressure?

Ppao is used most often in the bedside assessment of: (1) pulmonary edema, (2) pulmonary vasomotor tone, (3) intravascular volume status and LV preload, and (4) LV performance. These points were summarized recently and are restated below [25].

Pulmonary edema

Pulmonary edema can be caused by either elevations of pulmonary capillary pressure, referred to as hydrostatic or secondary pulmonary edema, or increased alveolar capillary or epithelial permeability, referred to as primary pulmonary edema. Usually hydrostatic pulmonary edema requires a pulmonary capillary increase to greater than 18 mm Hg; however, if capillary or alveolar cell injury is present, alveolar flooding can occur at much lower pulmonary capillary pressures. Furthermore, in the setting of chronic pulmonary vascular congestion, increased pulmonary lymphatic flow and increased respiratory excursions promote a rapid clearance of lung interstitial fluid, minimizing edema formation. Still, measures of Ppao are commonly used to determine the cause of pulmonary edema. Ppao values less than 18 mm Hg suggest a nonhydrostatic cause, whereas values greater than 20 mm Hg suggest a hydrostatic cause of pulmonary edema [22]; however, many exceptions to this rule exist. As mentioned above, if increased lung permeability is present, then fluid-resuscitation-induced pulmonary edema may occur at Ppao values much below 18 mm Hg, and treatment strategies aimed at reducing Ppao further will reduce pulmonary edema formation. Similarly, if pulmonary venous resistance is increased, then Ppc may be much higher than the measured Ppao, inducing hydrostatic pulmonary edema despite no increased lung permeability and a low Ppao. Similarly, Ppao may be greater than 20 mm Hg without any evidence of hydrostatic pulmonary edema, either because Ppl is also elevated or because of increased pulmonary lymphatic flow.

Pulmonary vasomotor tone

The pulmonary circulation normally has a low resistance, with pulmonary arterial diastolic pressure only slightly higher than Ppao and mean pulmonary arterial pressure a few mm Hg higher than Ppao. Pulmonary vascular resistance (PVR) can be estimated using Ohm's Law as the ratio of the pulmonary vascular pressure gradient (mean pulmonary artery

pressure minus Ppao) and cardiac output (ie, $PVR = (\text{mean Ppa} - \text{Ppao}) / \text{CO}$). Normal pulmonary vascular resistance is between 2 and 4 mm Hg \times l/min/m². Usually these values are multiplied by 80 to give normal pulmonary vascular resistance range in dynes sec/cm⁵ of 150 to 250. Either an increased pulmonary vascular resistance or a passive pressure buildup from the pulmonary veins can induce pulmonary hypertension. If pulmonary hypertension is associated with an increased PVR, then the causes are primarily within the lung. Diagnoses such as pulmonary embolism, pulmonary fibrosis, essential pulmonary hypertension, and pulmonary veno-occlusive disease need to be excluded. If PVR is normal, then LV dysfunction is the more likely cause of pulmonary hypertension [26]. Because the treatments for these two groups of diseases are quite different despite similar increases in pulmonary arterial pressure, the determination of PVR in the setting of pulmonary hypertension is very important. Regrettably, PVR poorly reflects true pulmonary vasomotor tone in lung disease states and during mechanical ventilation, especially with the application of PEEP. Alveolar pressure (Palv) can be the back pressure to pulmonary blood flow in certain lung regions during positive-pressure ventilation and in the presence of hyperinflation, because Palv exceeds left atrial pressure. Furthermore, because lung disease is usually nonhomogeneous, pulmonary blood flow is preferentially shifted from compressed vessels in West Zone 1 and 2 conditions (Ppao < Palv and Ppa = Palv, respectively) to those circuits with the lowest resistance (West Zone 3; ie, Ppao > Palv), thus making the lung vascular pathology appear less than it actually is.

Left ventricular preload

Ppao is often taken to reflect LV filling pressure, and by inference, LV end-diastolic volume. Patients who have cardiovascular insufficiency and a low Ppao are presumed to be hypovolemic and initially treated with fluid resuscitation, whereas patients who have similar presentations but an elevated Ppao are presumed to have impaired contractile function. Although there are no accepted high and low Ppao values for which LV underfilling is presumed to occur or not, Ppao values less than 10 mm Hg are usually used as presumed evidence of a low LV end-diastolic volume, whereas values greater than 18 mm Hg suggest a distended LV [27]. Unfortunately, there are very few data to support this approach. There are multiple documented reasons for this observed inaccuracy that relate to individual differences in LV diastolic compliance and contractile function [28]. First, the relation between Ppao and LV end-diastolic volume is curvilinear and is often very different among subjects and within subjects over time. Thus, neither absolute values of Ppao nor changes in Ppao will define a specific LV end-diastolic volume or its change [29]. Second, Ppao is not the distending pressure for LV filling. It is only the internal pressure of the pulmonary veins relative to atmospheric pressure. Assuming Ppao approximated left atrial

pressure, it will poorly reflect LV end-diastolic pressure, because it poorly follows the late diastolic pressure rise induced by atrial contraction and does not measure pericardial pressure, which is the outside pressure for LV distention. With lung distention, Ppl increases increasing pericardial pressure. Although we can estimate Ppl using esophageal balloon catheters, pericardial pressure is often different. Changes in pericardial pressure will alter LV end-diastolic volume independent of Ppao. Finally, even if one knew pericardial pressure and Ppao did accurately reflect LV end-diastolic pressure, LV diastolic compliance can vary rapidly, changing the relation between LV filling pressure and LV end-diastolic volume. Myocardial ischemia, arrhythmias, and acute RV dilation can all occur over a few heartbeats. Thus, is not surprising that Ppao is a very poor predictor of preload responsiveness. Therefore, it is not recommended to use Ppao to predict response to fluid resuscitation in critically ill patients.

Left ventricular performance

The four primary determinants of LV performance are preload (LV end-diastolic volume), afterload (maximal LV wall stress), heart rate, and contractility. Ppao is often used as a substitute for LV end-diastolic volume when constructing Starling curves (ie, relationship between changing LV preload and ejection phase indices). Usually one plots Ppao versus SWlv stroke work ($SVlv \times$ developed pressure). Using this construct, patients who have heart failure can be divided into four groups, depending on their Ppao ($>$ or <18 mm Hg) and cardiac index values ($>$ or <2.2 l/min/m²) [27]. Those patients who have low cardiac indices and high Ppao are presumed to have primary heart failure, and a low cardiac output and low Ppao, on the other hand, reflect hypovolemia. Those who have high cardiac indices and high Ppao are presumed to be volume overloaded, and having high cardiac output and low Ppao reflects increased sympathetic tone. Although this maybe a useful construct for determining diagnosis, treatment, and prognosis of patients who have acute coronary syndrome, it poorly predicts cardiovascular status in other patient groups. As described above, however, if LV end-diastolic volume and Ppao do not trend together in response to fluid loading or inotropic drug infusion, then inferences about LV contractility based on this Ppao/SWlv relation may be incorrect. This is not a minor point. Volume loading may induce acute RV dilation, markedly reducing LV diastolic compliance, such that Ppao will increase as SWlv decreases; however, the relationship between LV end-diastolic volume and swLV need not have changed at all. Similarly, inotropic drugs such as dobutamine may reduce biventricular volumes by decreasing venous return, decreasing LV diastolic compliance, even if the heart is not responsive to inotropic therapy. Thus, the same limitations on the use of Ppao in assessing LV preload must be considered when using it to assess LV performance.

Measuring cardiac output

Cardiac output can be estimated by many techniques, including invasive hemodynamic monitoring. Pulmonary blood flow using a balloon floatation PAC equipped with a distal thermistor, and transpulmonary blood flow using an arterial thermistor, both with a central venous cold volume injection, can be used. Similarly, minimally invasive echo Doppler techniques can measure blood flow at the aortic valve and descending aortic flow using esophageal Doppler monitoring. Cardiac output can be measured intermittently by bolus cold injection, or continuously by cold infusion. The advantage of the continuous cardiac output technique and the transpulmonary technique is that neither is influenced greatly by the ventilation-induced swings in pulmonary blood flow. Measurement of cardiac output by intermittent pulmonary artery flow measures using bolus cold indicator and monitoring the thermal decay curve is the most common method to measure cardiac output at the bedside; however, such intermittent measures will show profound ventilatory cycle-specific patterns [30]. By making numerous measures at random with the ventilatory cycle and then averaging all measures with proper thermal decay profiles, regardless of their values, one can derive an accurate measure of pulmonary blood flow [31].

Recently, a renewed interest in pulse contour analysis to estimate SVIv, and therefore cardiac output, from the arterial pressure profile over ejection has acquired its own set of supporters [32]. Arterial pressure and arterial pulse pressure are a function of rate of LV ejection, SVIv and the resistance, compliance, and inertance characteristics of the arterial tree and blood. If the arterial components of tone remain constant, then changes in pulse pressure most proportionally reflect changes in SVIv. Thus, it is not surprising that aortic flow variation parallels arterial pulse pressure variation [33], and pulse contour-derived estimates of stroke volume variation can be used to determine preload responsiveness [34,35]. Caution must be applied to using the pulse contour method, because it has not been validated in subjects who have rapidly changing arterial tone, as often occurs in subjects who have hemodynamic instability. Furthermore, it requires the application of abnormally large tidal volumes [34–36]. Thus, at the present time, the pulse contour-derived stroke volume variation technique represents a potentially great but still unproven clinical decision tool [37].

Currently three commercial devices that use pulse contour analysis of an arterial line waveform to obtain continuous cardiac output are approved for clinical use: PiCCO (Pulsion Medical Systems, Munich, Germany), LIDCO (Cambridge, United Kingdom), and Vigileo monitor (Edwards Lifesciences, Irvine, California) systems. Their benefit of being minimally invasive and the correlation shown with “standard” methods of measuring cardiac output in some clinical and experimental studies make them promising tools for hemodynamic monitoring [38,39].

Mixed venous oxygen saturation

Measuring venous oxygen saturation

SvO₂ reflects the pooled venous O₂ saturation, and is an important parameter in the assessment of the adequacy of DO₂ and its relation with VO₂. A decrease of SvO₂ could be explained by a decrease in DO₂ or any of the parameters that determine this, such as SaO₂, cardiac output, and hemoglobin concentration, and also by an increase in VO₂. A decrease of DO₂ will be followed by stable VO₂, with a consequent decrease of the SvO₂ until a critical value of DO₂ is reached where the tissues are no longer able to compensate having a constant VO₂, and VO₂ becomes dependent on DO₂ in an almost linear relation. At this level SvO₂, though continuing to decrease, becomes less sensitive to changes of tissue perfusion (Fig. 2).

SvO₂ measured from blood drawn from the distal tip of a PAC represents the true mixed venous value of the blood blended in the right ventricle. Care must be taken to withdraw blood slowly, so that it does not get aspirated from the downstream pulmonary capillaries. Validation of true mixed venous blood requires documentation that the measures PvCO₂ is greater than PaCO₂, because blood drawn over the capillaries sees alveolar gas twice, and will have a lower Pco₂ than arterial blood. Continuous measures of SvO₂ can be made using fiber optic reflectance spectroscopy. Two techniques are commercially available. Both use one fiber optic line to send a light signal and another to receive the reflected light at a different wavelength; however, only one catheter, Abbott, uses the Shaw technique of also measuring hemoglobin reflectance, and thus remains accurate over wide changes in hemoglobin concentration. The other catheter, Edwards, requires recalibration if hemoglobin levels vary by more than 1 gm/dL. Both techniques are valuable to monitoring SvO₂ trends as cardiac output, arterial O₂ content, or metabolic demand varies.

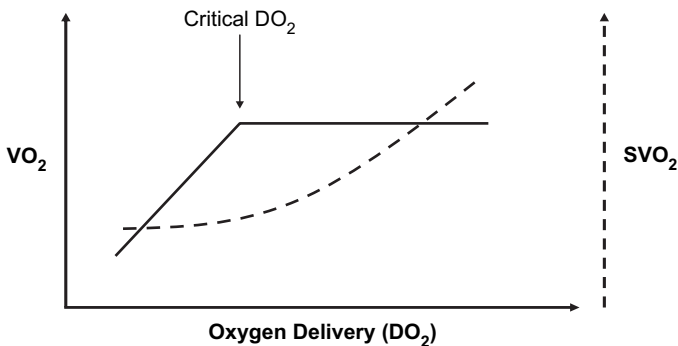


Fig. 2. This graph shows the interrelation of the determinants of SvO₂. The decrease of O₂ delivery under invariable oxygen consumption (VO₂) by the tissues will traduce a decrease of SvO₂, until a critical point where the consumption becomes dependent on the delivery almost in a linear relation; thus the SvO₂ in this case remains stable.

Superior vena caval oxygen saturation

Recent interest in superior vena caval O₂ saturation (ScvO₂) has evolved over the past years with the positive results of the Rivers and colleagues study [40], which demonstrated that in patients who had septic shock or severe sepsis admitted in the emergency department, an early and aggressive resuscitation guided by ScvO₂, CVP, and MAP reduced 28-day mortality from 46.5% to 30.5%. Measures of SvO₂, however, remain the gold standard to reflect minimal O₂ delivery. This is because although ScvO₂ and SvO₂ co-vary and seem to follow a parallel tracking, their differences can exceed 5%. Furthermore, during dynamic changes in cardiac output, such as occur in shock states, ScvO₂ may exceed SvO₂ by 5% or more, or be less than SvO₂ by 5% or more [41]. Thus, using a defined threshold value for ScvO₂ to identify when to start or stop resuscitation in a critically ill patient is fundamentally flawed. Still, a low ScvO₂ (<65%) is invariably associated with a low SvO₂ (<72%), making it less sensitive but still clinically useful at lower threshold values.

The meaning of cardiac output and venous oxygen saturation as end points of resuscitation

Although one may potentially measure cardiac output accurately at the bedside, there is no such thing as a normal cardiac output. Cardiac output is either adequate for the needs of the body or it is not. For example, the same cardiac output and DO₂ that is adequate at rest may be grossly inadequate and not associated with life during periods of increased metabolic demand. Because the primary goal of the cardiorespiratory system is to continuously maintain adequate amounts of O₂ (DO₂) to meet the metabolic demands of the tissues (VO₂), neither cardiac output nor mean arterial pressure are sensitive or specific measures of adequacy of cardiovascular function. Clearly, the best measures of adequacy of blood flow are the continued maintenance of normal end-organ function without evidence of excessive anaerobic metabolism. Normal urine output, gut activity, mentation, normal blood lactate levels, and spontaneous voluntary muscular activities reflect the most easily validated measures of body health [42]. Regrettably, many patients present with coexistent organ-system dysfunction, either pre-existent or caused by the insult. Furthermore, organ function cannot be monitored quickly enough to allow for titration of care. Thus, one cannot rely on these absolute markers to direct therapy [43]. Perhaps a more functional marker of adequacy of DO₂ to the tissues is SvO₂ [44]. Although values of SvO₂ greater than 70% do not ensure that all vascular beds are adequately perfused, SvO₂ values less than 60% are associated with oxidative impairment of tissues with a high metabolic rate, and values less than 50% are uniformly associated with evidence of anaerobic metabolism in some vascular beds [45]. Thus, as a negative predictive marker, preventing SvO₂ from decreasing below 50% and keeping it above

70% by fluid resuscitation, sedation, and ancillary support (eg, mechanical ventilation to reduce the work cost of breathing) all may improve O₂ delivery to metabolically active tissues.

If metabolic demand changes, cardiac output should co-vary with it [46]. Because this puts an added variable on the analysis of hemodynamic stability, a common approach in the cardiovascular management of the critically ill patient is to minimize the extraneous metabolic demands of the patient during intervals in which therapeutic interventions and diagnostic processes are being performed, so as to maintain stable baseline O₂ consumption for comparison. Thus, minimizing the work cost of breathing by using mechanical ventilation and reducing sympathetic responses by infusion of sedative agents reflect stabilizing processes that allow for accurate hemodynamic assessment. This is often more difficult to achieve than imagined [31]. Even a sedated and mechanically ventilated subject can be expending much effort assisting or resisting the ventilator-derived breaths. Muscular activities, such as moving in bed or being turned, “fighting the ventilator,” and breathing spontaneously can easily double resting VO₂ [47]. O₂ supply and demand must co-vary as a normal and expected aspect of homeostasis under almost all conditions. In cardiovascular insufficiency states, such as cardiogenic shock or hypovolemic shock, total cardiac output is often limited and cannot increase enough in response to increasing metabolic demand to match the demand. Under these severe conditions VO₂ tends to remain constant by varying the extraction of O₂ in the tissues rather than by varying total blood flow. Thus, measures of SvO₂ can be used to identify patients in circulatory shock. Furthermore, resuscitation efforts that increase SvO₂ to greater than 70% should be associated with improved end-organ function.

The controversy of the pulmonary artery catheter

One would think that the clinical use of the PAC in the management of the hemodynamically unstable patient would be invaluable; however, that utility has not been documented. Although currently there are no proven indications for the insertion of a PAC, there are potential indications (not yet proven) for its use based on the need to assess cardiac function, global DO₂, intravascular volume status, and pulmonary pressures, among others summarized in Box 4.

The controversy over the use of the PAC in the management of critically ill patients continues to rage. Proponents of its use cite a physiologic rationale to diagnosis and titration of complex treatments that may otherwise be detrimental. Opponents of its use cite the almost total lack of data showing that its use in the management of critically ill patients improves outcome. Still, one truth remains: no catheter will improve outcome unless coupled to a treatment that itself improves outcome.

Despite some exciting initial uncontrolled reports of markedly improved outcome in high-risk surgery patients [48,49], further well-controlled studies in both high-risk surgical patients [50] and trauma patients [51,52] failed to

Box 4. Probable indications for pulmonary arterial catheterization*Data necessary for diagnosis*

- Distinguishing primary (noncardiogenic) from secondary (cardiogenic) pulmonary edema
- Diagnosis of acute ventricular septal defect
- Diagnosis of acute cardiac tamponade

Data necessary for management

- Vasoactive drug therapy for cardiogenic shock with or without acute mitral regurgitation
- Cardiac dysfunction with ischemia requiring intra-aortic balloon counter-pulsation
- Balancing fluid and vasoactive therapy in acute lung injury states (acute respiratory distress syndrome [ARDS])
- Assessing pulmonary pathology and response to ventilator therapy in acute lung injury states
- To assess global cardiac output and systemic oxygen delivery
- To direct vasodilator therapy in the management of pulmonary hypertension associated with acute cor pulmonale
- To continuously monitor mixed venous oxygen saturation as an estimate of the adequacy of oxygen delivery to oxygen requirements in hemodynamically unstable patients

Adapted from Bellomo R, Pinsky MR. Invasive monitoring. In: Tinker J, Browne D, Sibbald W, editors. *Critical care: Standards, Audit and Ethics*. London: Hodder-Arnold, 1996; with permission.

document any improved survival when patients were treated based on pulmonary arterial catheter-derived data. In fact, the patients resuscitated aggressively to increase O₂ delivery into these survivor levels suffered a much higher mortality rate than did the control group treated conservatively [3]. Interestingly, as mentioned above, using only arterial pressure and ScvO₂, but with a defined physiology-based treatment algorithm, Rivers and coworkers [40] demonstrated a markedly improved survival in septic shock patients without the need of a PAC. On the other hand, a recent statistical analysis of the National Trauma Data Bank that included over 50,000 patients [53] showed for the first time a decrease in mortality in a selective group of trauma patients (severely injured, elderly, who arrived in shock) with the use of PAC.

Because of this unclear benefit on the use of PAC, the fact that it is an invasive monitoring procedure with potential serious complications acquires a major relevance when deciding about the risk-benefit of its use. Two recent

Box 5. Complications of the pulmonary artery catheter

Arrhythmia
Complete heart block
Catheter malpositioning
Extracardiac
Catheter knotting
Catheter fragmentation and meteorism
Pulmonary infarction
Pulmonary artery rupture
Thrombosis
Vascular infection

large prospective multicenter studies showed incidence of 5% and 10% of complications [54,55]. The most frequent complications described in these series were hematomas, arterial puncture, arrhythmias, and PAC-related infections, although a long list of complications has been described, as listed in [Box 5](#). No deaths attributable to PAC were found on this series, but other authors had previously reported mortality generally due to right heart and pulmonary artery perforation [56,57].

Beyond the controversial use of the PAC, two recent randomized clinical trials using active protocols of hemodynamic monitoring and algorithms of goal-directed therapy guided by esophageal Doppler flowmetry [58] and pulse contour analysis for cardiac output [59] in postoperative surgical patients showed a decreased duration of hospital stay and morbidity. Thus the literature suggests that the generalized use of hemodynamic monitoring and aggressive goal-directed therapy could improve outcome, but that one does not need to use a PAC to achieve these goals. These arguments, however, miss the point that the utility of hemodynamic monitoring is relative—no monitoring device, no matter how accurate, safe and simple to use, will improve outcome unless coupled to a treatment that itself improves outcome. Thus, the question should not be, “Does the PAC improve outcome?” but rather, “Do treatment protocols that require information only attainable from pulmonary arterial catheterization improve outcome?” Furthermore, the treatment protocol itself should also be shown to improve outcome, because otherwise if the trial shows no difference in outcome with or without a PAC, the results may well reflect the fact that there was no benefit for the protocol in either arm of the study.

Summary

All surgical patients require monitoring to assess cardiovascular stability, and sometimes may benefit from optimization of their hemodynamic status. Therefore, all surgeons require a basic understanding of physiological

underpinnings of hemodynamic monitoring. The physiological rationale is still the primary level of defense for monitoring critically ill patients.

Arterial catheterization to monitor arterial pressure is a safe procedure with a low complication rate; however, it should be used only when clear indications exist. There is no evidence that achieving pressures over 65 mm Hg increases organ perfusion or favors outcome. The analysis of pulse pressure variation is a useful method to assess preload responsiveness and a potential tool for resuscitation.

CVP has been wrongly used as a parameter of goal for replacement of intravascular volume in shock patients. Volume loading in patients who have CVP greater than 12 mmHg is unlikely to increase cardiac output, and attempts to normalize CVP in early goal-directed therapy during resuscitation have no proven benefit.

The use of PAC provides direct access to several physiological parameters, both as raw data and derived measurements (CO, SvO₂, DO₂). At present, targeting specific levels of DO₂ has proven effective only in high-risk surgery patients in the perioperative time. Ppao is often used as a bedside assessment of pulmonary edema, pulmonary vasomotor tone, intravascular volume status, and LV preload and performance. Several publications have explored the potential indications and benefits of the PAC to direct goal therapies. Beyond this controversy, there is a trend toward less invasive methods of hemodynamic monitoring, and current data support protocols of monitoring and goal-directed therapy that could improve outcome in selected group of surgical patients.

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