Interpretation of Hemodynamic Monitoring

Jonathan B. Mark, MD

Accurate interpretation of monitored hemodynamic values requires detailed understanding of the physiologic processes being measured and of the technical limitations of the monitoring procedures. This review course will focus on the most common invasive monitoring techniques, aiming to improve the clinical utility of these procedures through highlighting the common problems in arterial, central venous, and pulmonary artery pressure monitoring.

Blood Pressure: Why Don't the Numbers Agree?

A patient arrives in the intensive care unit after vascular surgery, with a left radial artery catheter blood pressure reading of 180/65 mm Hg and a right arm noninvasive cuff pressure reading of 135/75 mm Hg. Which is correct? Which values should be used to guide patient treatment?

This clinical scenario is common and presents very practical issues for the physician managing this patient. Both indirect noninvasive and direct invasive blood pressure (BP) measurements may yield inaccurate results, owing to a number of patient factors and technical considerations. Standard indirect measurements of blood pressure are performed most commonly with automated devices that use an oscillometric technique for noninvasive blood pressure (NIBP) measurement. Arterial pulsations are sensed by the monitor through the partially occlusive cuff. The mean arterial pressure is the pressure at which maximal oscillations are detected and the systolic and diastolic pressure values are determined at pressures where the pressure oscillations begin and disappear. The standard auscultatory method using a blood pressure cuff and stethoscope identifies systolic and diastolic pressures at the points of onset and disappearance of the sounds of turbulent blood flow (Korotkoff Sounds). Although the oscillometric and auscultatory methods provide similar values for blood pressure in most patients, they are inherently different techniques, and prone to error or failure under certain conditions. These include the following:

- Impaired acoustic transmission (Auscultatory).
- Extrinsic cuff compression (Auscultatory, NIBP).
- Overly rapid cuff deflation leading to BP underestimation (Auscultatory).
- Calcified, noncompressible arteries leading to BP overestimation (Auscultatory, NIBP).
- Intense vasoconstriction leading to BP underestimation (Auscultatory).
- Use of inappropriately small cuff leading to BP overestimation (Auscultatory, NIBP).
- Dysrhythmias (Auscultatory, NIBP).
- Shivering and patient movement (Auscultatory, NIBP).
- Beat-to-beat BP variations, as in pulsus alternans (Auscultatory, NIBP).
- Rapid BP changes not detected (Auscultatory, NIBP).

Direct invasive blood pressure measurement with an arterial catheter is commonly used when rapid changes in blood pressure are anticipated or repeated blood sampling is needed. As in the case for noninvasive indirect BP measurement, technical factors influence the accuracy of direct arterial BP measurement also. The primary factor that must be appreciated is that the arterial pressure waveform changes as it is transmitted from the aortic root to the peripheral arteries where it is commonly measured. The arterial pressure waveform undergoes distal pulse amplification owing to the impedance and harmonic resonance of the vascular tree. Compared with a central aortic pressure trace, a radial artery pressure waveform displays a delayed but steeper upstroke, higher systolic peak, delayed dicrotic notch, exaggerated diastolic wave, and lower end-diastolic pressure. In other words, the arterial pulse pressure is wider in the periphery, and the monitored values of systolic and diastolic pressure will be higher and lower, respectively, than the simultaneous values measured in the central aorta. Fortunately, mean arterial pressure changes little as the arterial waveform is transmitted to the periphery.

Technical considerations also influence the accuracy of direct arterial BP measurement. The accuracy of the monitored arterial BP waveform depends on the physical characteristics of the monitoring system (e.g., catheter, tubing, stopcocks), which determine the two key

parameters of system performance, the natural frequency and damping coefficient. Most simply, the natural frequency describes how rapidly the arterial monitoring system oscillates, and the damping coefficient describes how quickly it comes to rest. When the arterial pressure monitoring system has too low a natural frequency and is underdamped (conditions that are often present in clinical practice), the system resonate or "rings." This pressure measurement artifact, often termed "overshoot," results in factitiously elevated systolic BP values and is commonly seen in patients with tachycardia and steep arterial systolic pressure upstrokes because of the frequency content of these pressure waveforms.

Given the technical considerations underlying both indirect noninvasive and direct arterial BP measurement, it is no surprise that different methods of BP measurement provide different values. In the perioperative care of critically ill patients, a host of additional factors further contribute to these pressure differences. Patients with peripheral vascular disease commonly have regional arteriosclerosis that manifests as BP differences in excess of 20 mm Hg between right and left arms. Any palpable difference in right and left arm radial pulses mandates cuff pressure measurements in both arms to determine the magnitude of the difference and plan an appropriate site for arterial catheterization.

More generalized arterial pressure gradients frequently appear in certain clinical circumstances, including immediately after cardiopulmonary bypass and vasodilatory shock states. In these situations, the normal distal pulse wave amplification is not observed. In contrast, peripheral arterial BP measurements markedly underestimate the central aortic BP. The mechanism underlying these vascular phenomena involves relative proximal arterial vasoconstriction in combination with profound peripheral arteriolar vasodilation.

In summary, blood pressure is a function of measurement site and technique. Recognition of confounding technical and patient-specific factors is required to avoid measurement errors and misguided therapy.

Central Venous Pressure: Where Should the Transducer be Positioned?

A 68-yr old patient scheduled for a Whipple procedure undergoes central venous cannulation for intraoperative monitoring. The central venous pressure reads 13 mm Hg. There is no history of cardiac or pulmonary disease. Is this patient hypovolemic, euolemic, or hypervolemic?

Before monitoring any intravascular pressure, the external transducer is "zeroed" and adjusted to the appropriate "level" on the body. Though often performed at the same time, these two actions are independent. The transducer is zeroed by exposing it to ambient atmospheric pressure, usually by opening an attached stopcock, and then pressing the zero pressure control on the bedside monitor. The transducer now has a standard reference value, ambient atmospheric pressure, which is assigned the value of 0 mm Hg and used as the reference point for all subsequent intravascular pressure measurements. During all hemodynamic monitoring procedures, the displayed pressures are referenced to ambient atmospheric pressure in the room outside the body. Regardless of where the transducer is placed, the zero reference value should not change, and the zeroing procedure should only be repeated if the reference value has drifted from 0 mm Hg.

The second step in transducer setup involves placing the transducer at the appropriate vertical height relative to the patient's position. Typically, in a supine patient, the transducer is aligned with the midchest position in the midaxillary line. This traditional site is easy to estimate by eye and provides a reasonable estimate of the location of the heart within the thorax, but is it the appropriate position for central venous and other central vascular pressure monitoring?

Central venous pressure (CVP) is measured as an estimate of cardiac preload, specifically right ventricular preload. The force that determines preload or chamber volume is the distending pressure across the wall of the right atrium (and, at end-diastole, the right ventricle). The confounding effect of the "weight" of the blood within the chamber is not of interest, only the transmural pressure that distends the chamber. As a result, external pressure transducers should be placed in a position that obviates this confounding effect of the hydrostatic pressure effect of the blood within the cardiac chamber. This is best done by aligning external pressure transducers with the uppermost blood level in the chamber from which pressure is being measured. Based on echocardiographic data, the best transducer placement for standard clinical monitoring is at a vertical height approximately 5 cm below the left sternal border at the fourth intercostal space. The common clinical practice of transducer alignment at the midchest level results in significant pressure overestimation, more than 5 mm Hg in the average patient.

Transducer positioning at the level described above, 5 cm below the sternal border, identifies the uppermost level of the left ventricular cavity. Fortunately, the upper levels of blood in the right ventricle, right atrium, and left atrium are all within approximately 1 cm (i.e., $\pm$ 0.8 mm Hg) and, consequently, this position is an appropriate transducer height for measurement of both right heart (CVP) and left heart (pulmonary artery wedge pressure) filling pressures.

Pulmonary Artery Wedge Pressure: What Is It, and What Does It Mean?

When a pulmonary artery catheter (PAC) is advanced to the wedge position, the inflated balloon isolates the pressure-measuring orifice at the catheter tip from upstream pulmonary artery pressure. A continuous static column of blood now connects the catheter tip in the pulmonary artery with a junction point where flow resumes in a pulmonary vein near its entrance to the left atrium. Under these conditions, there is no flow between the catheter tip and this venous junction point. Consequently, obtaining the pulmonary artery wedge pressure (PAWP) functionally extends the PAC tip into the left atrium and provides an indirect measurement of left atrial and left ventricular diastolic pressure. Measurement of PAWP requires this uninterrupted column of blood across the pulmonary vascular bed. When alveolar pressure is high or left atrial pressure is low, these physiologic conditions may not be present, and the PAC will inappropriately measure alveolar pressures rather than downstream left atrial pressure.

Wedge pressure waveforms mirror left atrial pressure waveforms, just as CVP waveforms mirror right atrial pressure waveforms. However, because PAWP is an indirect upstream measurement of left atrial pressure, the normal phasic left atrial pressure waves will appear to be delayed when recorded in the PAWP tracing. The a wave seen in a normal CVP tracing, which results from end-diastolic right atrial contraction, appears after the ECG P wave and before the ECG R wave. By analogy, the a wave seen in a normal PAWP tracing results from end-diastolic left atrial contraction. However, owing to the 150–200 ms delay in transmission of the left atrial pressure waves back through the pulmonary vascular bed, the PAWP a wave will be seen to follow the ECG R wave in most patients and thereby appear to be a cardiac cycle event occurring in early ventricular systole. The temporal delay between left atrial pressure waves and their indirect recording in the PAWP must be kept clearly in mind to avoid confusion of a PAWP a wave for a v wave and the resulting misdiagnosis and inappropriate treatment.

Like CVP measurement, PAWP is measured as a surrogate for cardiac preload, more specifically left ventricular preload. As left ventricular end-diastolic volume increases, so too does end-diastolic pressure, which is reflected as an increase in PAWP. However, there are two confounding factors that must always be considered. First, filling volume of any cardiac chamber is a function of transmural distending pressure, the difference between the pressure inside and outside the chamber. However, all intravascular filling pressures are referenced to ambient atmospheric pressure, which was assigned a value of 0 mm Hg at the beginning of the monitoring period. As intrathoracic and intrapericardial pressure increases, transmural chamber pressure may fall even though the transduced pressure inside the cardiac chamber increases. The cardiac chamber is smaller even though the measured intravascular pressure has increased. Chamber size (cardiac muscle fiber length or preload) is determined by transmural pressure, not simply transduced pressure. Commonly, transmural filling pressure and cardiac chamber size are reduced by increased intrathoracic pressure (positive end-expiratory pressure) or by increased intrapericardial pressure (cardiac tamponade). The increased PAWP recorded in these circumstances belies the fact that left ventricular volume is normal or decreased.

Altered diastolic ventricular compliance also confounds interpretation of the PAWP as a surrogate for left ventricular preload. The diastolic pressure-volume relation of the left ventricle is curvilinear, with minimal pressure changes at low volume and large pressure changes at the limits of compliance. Conditions that induce diastolic dysfunction and impair ventricular compliance, such as left ventricular hypertrophy and myocardial ischemia, will be clinically manifest as an increase in PAWP at any given level of ventricular volume.

Hemodynamic Monitoring: Who, When, Why?

The controversy surrounding use of the PAC (1–4) was re-ignited by the controversial study published by Connors et al. in 1996 (5). This 5,735-patient prospective cohort study conducted in critically ill patients in the intensive care unit described excess cost, length of stay, and mortality associated with use of the PAC. Independent experts and consensus panels suggested strongly that there is tremendous variability in the hemodynamic monitoring skills and knowledge among users of the PAC, and that failure to demonstrate improved outcome with PACs derives in part from misuse of the PAC (6–16). How can this problem be addressed? Multiple educational sources are available, including practice guidelines offered by the major medical societies. The 1993 American Society of Anesthesiologists (ASA) Task Force on Pulmonary Artery Catheterization recently reconvened to update its practice parameter on PAC use. The ASA House of Delegates approved this document in October 2002, and it will be published in 2003.

Other educational sources abound, including the Pulmonary Artery Catheter Education Project (PACEP), an outgrowth of nationally funded PAC workgroups. The PACEP website (http://www.pacep.org) offers an opportunity for all clinicians to acquire online education and CME credits. What better way to improve your skills in interpretation of hemodynamic monitoring?

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