Monitoring Arterial Blood Pressure: What You May Not Know

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Arterial blood pressure (ABP) is a basic hemodynamic index often utilized to guide therapeutic interventions, especially in critically ill patients. Inaccurate ABP measuring creates a potential for misdiagnosis and mismanagement. The results of a recent pilot study* at 2 university-affiliated hospitals suggested a knowledge deficit in arterial pressure monitoring and some of the most basic aspects of hemodynamic monitoring. A total of 391 critical care nurses practicing in various critical care specialties were invited to participate in the study. The response rate was 17.4% (n = 68). Most of the participants were between the ages of 30 and 39 years (56.1%) and had a baccalaureate degree as their basic (61.8%) and highest degree in nursing (58.8%). Most participants had more than 4 years of nursing experience (94.1%) and critical care experience (83.9%), and most did direct ABP monitoring at least once or twice each week (97.1%). The participants were asked to complete an 18-item, criterion-referenced questionnaire on ABP physiology, physiological and pathophysiological factors that affect ABP, and the arterial pressure waveform and its interpretation in clinical situations common in critical care patients.

ABP PHYSIOLOGY
The cardiovascular system has 3 types of pressures\(^6,7\): hemodynamic, kinetic energy, and hydrostatic. Hemodynamic pressure is the energy imparted to the blood by contraction of the left ventricle. This type of pressure is preserved by the elastic properties of the arterial system. Kinetic energy is the energy associated with motion and affects the pressure measured during direct ABP monitoring. Fluid density and gravity contribute to hydrostatic pressure, which is the pressure a column of fluid exerts on the container wall. For example, in a column of fluid, the pressure at a given level in the container is proportional to the

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height of the fluid column above that level. The pressure is highest at the bottom of the column. In the vascular system, hydrostatic pressure is proportional to the height of the column of blood between the heart and the peripheral vasculature. In a standing person, the pressure in the leg is higher than the pressure in the arm by the difference in hydrostatic pressure. In summary, arterial blood pressure represents the force exerted by the blood per unit area on the arterial wall and is the sum of hemodynamic, kinetic, and hydrostatic pressure.

The arterial tree starts with the aorta and the major branches of this vessel. The aorta and its branches stretch to receive blood from the left ventricle and recoil to distribute the blood and to maintain arterial pressure. Arteries and arterioles control blood pressure through vasoconstriction or vasodilation. Arterioles are the primary sites that contribute to systemic vascular resistance (SVR). In addition, adrenergic control of the arterioles is a major determinant of blood flow into the capillaries. In the skin, for instance, blood can be shunted from the capillary beds to flow directly from arterioles into the venous system. Arteriovenous shunting occurs in shock states and helps to redirect blood flow to vital organs. Arteriovenous shunting is one reason measurements of blood pressure alone are not a good indicator of peripheral tissue perfusion.

Arterial pressure is measured at its peak, which is the systolic blood pressure (SBP), and at its trough, which is the diastolic blood pressure (DBP). The SBP is determined by the stroke volume, the velocity of left ventricular ejection (an indirect indicator of left ventricular contractile force), systemic arterial resistance, the distensibility of the aortic and arterial walls, the viscosity of blood, and the left ventricular preload (end-diastolic volume). The blood pressure in the aorta during systole is a clinical indicator of afterload (the sum of the forces the left ventricle must overcome to eject blood). The diastolic pressure is affected by blood viscosity, arterial distensibility, systemic resistance, and the length of the cardiac cycle.

Pulse pressure is the difference between systolic and diastolic pressure. A normal pulse pressure in the brachial artery is approximately 40 mm Hg. An increased pulse pressure may be the result of increased stroke volume or ejection velocity and is common during fever, exercise, anemia, and hyperthyroidism. Other causes of increased pulse pressure include bradycardia (increased stroke volume), aortic regurgitation, and arterial stiffening, which is most noticeable after the age of 50 to 60 years. An acute decrease in pulse pressure may indicate an increase in vascular resistance, decreased stroke volume, or decreased intravascular volume.

Systemic mean arterial pressure (MAP) is defined as the mean perfusion pressure throughout the cardiac cycle. MAP is sensed by baroreceptors located in the carotid sinuses and the arch of the aorta. These receptors control arterial pressure mainly by adjusting heart rate and arteriolar vessel radius. MAP is also the basis for autoregulation by some organ systems such as the kidney, heart, and brain. Autoregulation is the automatic adaptation of the radius of an arteriolar vessel in an organ to maintain constant blood flow over a wide range of mean pressures (60-150 mm Hg) to protect functioning of the organ. MAP is the product of SVR and cardiac output (MAP = SVR x cardiac output).

As indicated previously, a main determinant of SVR is the radius of arterial, and particularly arteriolar, vessels. Changes in cardiac output are related to heart rate and stroke volume. Stroke volume, in turn, is determined by several factors, including heart rate, preload, afterload, cardiac contractility, and energy of cardiac contraction (related to ventricular dilatation, abnormalities in ventricular wall motion, and ventricular arrhythmias). MAP is generally closer to diastolic pressure because diastole represents about two thirds of the cardiac cycle when the mean heart rate is close to 60/min. This relationship is expressed in the well-known formulas MAP = DBP + (SBP - DBP)/3 and MAP = [SBP + (DBP x 2)]/3.

However, the proportion of diastole in the cardiac cycle changes with changes in heart rate. In calculations of MAP for a manually obtained ABP, these formulas must be used with caution, because they provide a good estimate of MAP only when the heart rate is close to 60/min. Fortunately, MAP is provided by most automatic ABP measuring devices and direct ABP monitoring systems, each of which uses a systemspecific method to directly determine MAP.

In summary, because of the multiplicity of factors that contribute to ABP and the complexity of their interrelationships, interpreting changes in arterial pressure and its components (SBP, DBP, MAP, and pulse pressure) as indicative of any single factor may lead to an erroneous assessment of a patient’s condition. When SBP
and DBP are measured using different (oscillometric or direct) monitoring methods, the values can differ significantly. When MAP is measured using different monitoring methods, however, the values are very similar, because MAP is little affected by the phenomenon of wave reflection.21-25 Wave reflection and other factors that affect measurement of SBP and DBP are discussed later.

**PRINCIPLES OF HEMODYNAMIC MONITORING**

Three Conventions of Cardiovascular Pressure Measurement

For measurement of cardiovascular pressures, 3 conventions are observed6,7,20: (1) Cardiovascular pressures are expressed in millimeters of mercury, with the exception of central venous pressure, which may be measured in millimeters of mercury or in centimeters of water. For converting values in centimeters of water to values in millimeters of mercury, the value given in centimeters of water is divided by the factor 1.36. (2) Most cardiovascular pressures, such as ABP, central venous pressure, and pulmonary artery pressure, are referenced to the heart or, more specifically, to the atria, to eliminate hydrostatic pressure. (3) All cardiovascular pressure monitoring devices are zeroed to ambient atmospheric pressure, so that the actual pressure measured reflects the pressure above atmospheric pressure.

The Hemodynamic Monitoring System

A hemodynamic monitoring system contains 2 compartments: the electronic system and the fluid-filled tubing system. Although clinicians have little control over the electronic components such as the monitor, correct setup and maintenance of the tubing system and the pressure transducer are absolutely crucial to avoid error. With an improperly prepared or inadequately functioning monitoring system, not only the actively measured hemodynamic indices but also any derived variables will be erroneous,29 potentially invalidating a patient’s entire hemodynamic profile. Three procedural steps should be followed to prepare the monitoring tubing system and ensure its continued accuracy: priming of the pressure tubing, leveling and zeroing, and dynamic response testing.

*Priming of the Pressure Tubing.* The generation and recording of all arterial waveforms (systemic arterial pressure and pulmonary artery pressure) are based on the same basic principles. The invasive catheter provides access to the arterial system being monitored and is designed to pick up the pressure waves generated in the arterial system by cardiac contractions. The catheter is connected to the fluid-filled tubing of the monitoring system. The fluid column in the tubing system carries the mechanical signal created by the pressure wave to the diaphragm of the electrical pressure transducer. The transducer creates the link between the fluid-filled tubing system and the electronic system, and converts the mechanical signal into an electrical signal. The electrical signal is transmitted to the monitor and then is amplified and displayed as an analog waveform and digital output.

Air as a medium transmits mechanical impulses much differently than does fluid. Air bubbles in the tubing system are one of the most frequent and important sources of error in hemodynamic monitoring. Air bubbles most often blunt or damp propagation of the mechanical signal, causing a damped analog waveform and erroneous pressure readings.27 Even tiny air bubbles only 1 mm in diameter can cause serious waveform distortion.28 Therefore, air-free priming of the entire tubing system is one of the most important steps to avoid technical error.

Air-free priming starts with removal of all air from the flush solution to prevent air from going into the solution as a result of the pressure applied by the external pressure cuff. Then, the entire tubing system should be flushed. Stopcocks, Luer-Lok interconnections, and the transducer are common locations of air entrapment27 and deserve special attention throughout priming and use of the catheter system. In order to maintain the air-free status after the initial setup of the system, the following measures are important:

- After opening the system for blood sampling or zeroing, briefly fast-flush the tubing system, including the proximal access stopcock or the air-fluid interface stopcock.
- Tighten all connections, and ensure that the stopcocks are closed to air.
- Avoid adding stopcocks and line extensions.
- Keep the flush solution bag adequately filled, and keep the external pressure cuff at 300 mm Hg.
- Periodically flick the tubing system and flush the tubing system and stopcocks to eliminate tiny air bubbles escaping the flushing solution.

*Leveling and Zeroing.* The arterial pressure monitoring sys-
tem must be referenced to heart level, technically the level of the left atrium, and set at atmospheric pressure as the zero reference point. These criteria can be met through leveling and zeroing. Leveling or referencing of the catheter system is accomplished by aligning the air-fluid interface of the monitoring system (eg, the stopcock on top of the transducer) with the external reference point of the heart. The external reference point of the heart is the phlebostatic axis, which can be located by finding the junction of 2 lines: a vertical line drawn out from the fourth intercostal space at the sternum and a horizontal line drawn through the midpoint of a line going from the anterior to the posterior side of the chest.29,30

Where to level the air-fluid interface is a matter of discussion. The answer depends on which vascular bed is to be monitored. In most clinical situations, the central arterial pressure is the pressure of interest, because it is a key determinant in cardiac and cerebral perfusion. In order to monitor central arterial pressure, the monitoring system must be leveled to the heart by using the phlebostatic axis. Research indicates that the phlebostatic axis most accurately reflects the level of the atrium in both supine and upright patients.29-31 The midaxillary line, which has also been used as an external reference point for the heart, is not accurate in all chest configurations and thus is not recommended.31 When the monitoring system is referenced to the tip of the catheter, then the transmural pressure of a particular point in the arterial tree is monitored and not central arterial pressure. Peripherally measured transmural pressure is markedly increased by hydrostatic pressure unless the patient is supine.32

Zeroing consists of 2 steps. First, the air-fluid interface is opened to atmospheric pressure. Then the monitor’s zeroing function key or button is pressed. Zeroing in this fashion has several purposes. Zeroing electronically establishes for the monitor atmospheric pressure as the atmospheric zero reference point. Zeroing establishes the interface level as the hydrostatic zero reference point. Zeroing also eliminates zero-drift. Zero-drift is the potential, but usually minimal, transducer offset or distortion occurring over time.33

Two key practice objectives are related to referencing and zeroing: accuracy and consistency. In order to ensure accuracy, leveling and zeroing must be done whenever the relationship between the air-fluid interface and the reference point is changed. The reason is hydrostatic pressure. For every 1 cm the air-fluid interface is above or below the actual level of the left
atrium, 0.74 mm Hg of hydrostatic pressure is subtracted or added to the measured pressure. If the air-fluid interface is placed 10 cm below the phlebostatic axis, the measured pressure will be an over-estimation of the actual hemodynamic pressure by 7.4 mm Hg. This example illustrates that though accurate referencing and zeroing are important for all hemodynamic monitoring, they become even more crucial when small hemodynamic pressures, such as pulmonary artery wedge pressure (6-12 mm Hg) and central venous pressure (2-6 mm Hg) are being monitored. In these instances, small offsets from the phlebostatic axis and the zero reference point can cause large errors and may prompt inappropriate treatment.

In order to ensure consistency, once the external reference point has been selected, it should be marked on the patient for easy identification. Consistency allows correct determination of trends in a patient’s hemodynamic status. Whether the same body position must be used for consecutive measurements may be debatable. Although several studies on pulmonary artery pressure monitoring indicated no significant changes in pulmonary artery pressures related to certain bed positions, according to a recent research abstract, ABP values obtained with various bed positions differed even when the phlebostatic axis was used as the external reference point. The statistical significance of differences in ABP values for monitoring systems referenced to the phlebostatic axis was not addressed in the abstract.

Dynamic Response Testing. In order to determine if a hemodynamic monitoring system can adequately reproduce a patient’s cardiovascular pressures, the dynamic response characteristics of the catheter system must be tested. Only when system accuracy, also termed fidelity, has been confirmed, can the analog waveform be accepted as an accurate reflection of a patient’s status.

The dynamic response of a hemodynamic monitoring system is defined by its natural frequency and the damping coefficient. The natural frequency indicates how fast the pressure monitoring system vibrates when shock excited by a signal such as the arterial pressure pulse or the pressure signal caused by a fast-flush test. The damping coefficient of a monitoring system is a measure of how quickly the oscillations of a shock-excited system dampen and eventually come to rest.

Dynamic response testing is a 3-step procedure: determining natural frequency, determining the amplitude ratio of 2 consecutive fast-flush oscillations (as an indirect way of determining the
damping coefficient), and determining the dynamic response characteristics of the monitoring system (eg, optimal, adequate, underdamped, overdamped, or unacceptable). These steps are summarized in Figure 1.

Dynamic response testing may seem complicated at first. Clinical experience has shown, however, that with proper training, dynamic response testing can be performed in less than 2 minutes. A few simple observations provide almost as much information and may serve as a close estimate. (1) If the period between 2 oscillations of the fast-flush test is less than 1.2 mm, then natural frequency will be at least 21, and the system will most likely be adequate. If the period between 2 oscillations is 1 mm or less, then the natural frequency is 25 or higher, and the system will almost always function properly with any degree of damping. As a rule-of-thumb, the higher the natural frequency (or the smaller the period), the better is the dynamic response of the monitoring system. (2) The system is overdamped if the fast-flush produces sluggish or no oscillations. If the square wave shows undulations or if ringing occurs after the release of the fast-flush device, the system is most likely underdamped. Overdamping and underdamping both indicate that the dynamic response characteristics of the monitoring system are unsatisfactory.

Simple visual evaluation of the arterial waveform, the square waveform, and oscillations generated by the fast-flush has been suggested as a suitable method for determining the dynamic response characteristics of a monitoring system. However, the square waveform and fast-flush oscillations may look adequate when the natural frequency and amplitude ratio of the system actually make the system inadequate (underdamped, overdamped, or unacceptable). Or, the arterial waveform may look distorted because of physiological variations, yet the dynamic response characteristics of the system assure a faithful recording of the arterial pressure. When accuracy is needed for clinical decision making, the ideal method of determining if the measured ABP, or any other directly monitored hemodynamic parameter, is true is to determine the dynamic response characteristics of the monitoring system. Examples of the importance of formally assessing the response characteristics are discussed in more detail later.

COMPONENTS OF THE NORMAL ABP WAVEFORM

Left ventricular contraction creates a pressure pulse or pulse wave. It is the pressure pulse that a clinician feels when determining a patient’s pulse by palpation. The pressure pulse is also what is sensed by the intra-arterial catheter. The normal arterial pressure waveform is shown in Figure 2. The systolic upstroke or anacrotic limb mainly reflects the pressure pulse produced by left ventricular contraction. The pressure pulse is followed slightly later by the flow wave caused by the actual displacement of blood volume. The anacrotic shoulder, that is, the rounded part at the top of the waveform, reflects primarily volume displacement. The systolic pressure is measured at the peak of the waveform. The dicrotic (or downward) limb is demarcated by the dicrotic notch, representing closure of the aortic valve and subsequent retrograde flow. The location of the dicrotic notch varies according to the timing of aortic closure in the cardiac cycle. For example, aortic closure is delayed in patients with hypovolemia. Consequently, the dicrotic notch occurs farther down on the dicrotic limb in hypovolemic patients. The dicrotic notch also appears lower on the dicrotic limb when arterial pressure is measured at more distal sites in the arterial tree (Figure 3). The shape and proportion of the diastolic runoff wave that follows the dicrotic notch changes with arterial compliance and heart rate. Diastolic pressure is measured just before the beginning of the next systolic upstroke.

The analog waveform visible on the monitor or recorded on a strip chart is not only caused by the forward pressure pulse but is also a result of a phenomenon known as wave reflection. Wave reflection is related to the impedance of blood flow by the narrowing and bifurcation of the arterial vessels; the impedance leads to backward or retrograde reflection of the pressure wave. In a manner similar to waves on the beach, the forward or antegrade pressure waves and the reflected waves collide. The combination of the 2 types of waves increasingly augments the SBP the farther down the blood pressure is measured in the arterial circuit. In other words, the contribution of reflected waves to the measured systolic pressure occurs earlier in the periphery, particularly in the radial and dorsalis pedis arteries (Figure 3), where the measured SBP may be 20 to 25 mm Hg higher than central aortic
1. Determining natural frequency (Fn)
   a. Perform the fast-flush maneuver (square waveform test): pull and release the pigtail or compress and release the button of the fast-flush device of the monitoring system.
   b. Record the resulting square waveform and subsequent oscillations on calibrated strip chart paper.
   c. Measure the distance (period, t, of 1 cycle) in millimeters between 2 oscillations. (One small box on the calibrated strip chart paper equals 1 mm.)
   d. Calculate Fn by using the formula
      \[ Fn = \frac{\text{paper speed (mm/s)}}{t \text{ of 1 cycle (mm)}} \]
      (The standard paper speed is 25 mm/s.)
      Example: paper speed = 25 mm/s; t = 1 mm; \( Fn = \frac{25 \text{ mm/s}}{1 \text{ mm}} = 25 \text{ Hz} \)

2. Determine the amplitude ratio
   a. Measure the amplitude (A) in millimeters of 2 successive oscillations (A1, A2).

3. Determine the dynamic response characteristics
   a. Plot Fn along the x-axis (result of step 1)
   b. Plot the amplitude ratio along the y-axis on the right (result of step 2)
   c. Find the intersection of the 2 lines. Where the 2 lines intersect on the Fn-versus-amplitude ratio graph determines if the system is able to correctly reproduce the hemodynamic waveform (adequate, optimal) or if the system is not functioning at a desirable level (overdamped, underdamped, unacceptable).

Figure 1 The 3 steps of dynamic response testing. A, The fast-flush test. B, The frequency-versus-damping coefficient graph.
A, Adapted from Bridges and Middleton," with permission. B, Reprinted from Gardner and Hollingsworth,28 with permission; adapted from Bridges and Middleton," with permission.
pressure.\textsuperscript{22,23} Under normal conditions, 80\% of the original wave is thought to be reflected.\textsuperscript{22}

Clinically, wave reflection plays an important role in left ventricular workload and cardiac oxygen consumption. In young adults with elastic arteries, the reflected wave returns to the heart during the diastolic phase of the cardiac cycle and thus augments coronary artery perfusion. In elderly patients or patients with stiff, atherosclerotic vessels, the reflected wave returns to the heart during systole and thus increases systolic pressure and left ventricular afterload.\textsuperscript{39}

Pharmacological vasoconstriction can similarly increase wave reflection and cardiac workload.\textsuperscript{23,39}

The contribution of reflected waves to the measured systolic pressure is diminished during hypovolemia, hypotension, the Valsalva maneuver, and vasodilatation.\textsuperscript{21} In pharmacologically induced vasodilation, such as occurs with nitroglycerin for instance, peripherally measured systolic pressure may not change in proportion to the actual degree of reduction in central aortic pressure.\textsuperscript{38,40} The effect of nitroglycerin may be visible in the appearance of reflected waves after the systolic peak (Figure 4), but reduced aortic pressure, afterload, and cardiac work load may be more evident through clinical improvement of the patient. During shock with vasoconstriction, wave reflection can lead to the overestimation of central aortic pressure, because peripheral SBP may be 20 mm Hg higher than aortic pressure.\textsuperscript{36,39} Peripherally measured SBP could in this situation provide a false sense of security that the patient is maintaining adequate perfusion pressures. A slower systolic upstroke and a prominent diastolic waveform with reflected waves may be visual indicators of shock with vasoconstriction\textsuperscript{39} (Figure 5).

A common scenario when working with arterial catheters is comparing the blood pressure recorded by the arterial catheter with the blood pressure obtained manually or with an oscilloscope. If a discrepancy occurs, the arterial catheter is often said to be somehow “damped,” with the underlying understanding that pressure readings obtained with the catheter cannot be trusted. Such an interpretation may be premature and probably disregards several important facts. Differences exist between (1) damping, overdamping, and underdamping; (2) overdamped and underdamped pressure waveforms caused by overdamped or underdamped monitoring systems and overdamped or underdamped-appearing waveforms that reflect the true physiological status of a patient; and (3) blood pressures obtained via direct versus indirect monitoring methods.

All hemodynamic monitoring systems are damped. Damping is desired, because without damping the vibrations of the system’s fluid column caused by the arterial pressure pulse would go on indefinitely and no accurate waveform could be recorded. Conversely, both overdamped and underdamped systems exist, and their recordings are indeed erroneous. With an overdamped system, the waveform loses its characteristic landmarks and

**Figure 2** The normal arterial pressure waveform. MAP indicates mean arterial pressure. Reprinted from Darovic,\textsuperscript{10} with permission.

**Figure 3** Changes of the arterial pressure waveform configuration throughout the arterial tree. Note the increasing steepness and amplitude of the systolic upstroke and the changing location of the dicrotic notch. Reprinted from Gornon,\textsuperscript{1} with permission.

**Figure 4** Changes of the arterial pressure waveform configuration throughout the arterial tree. Note the increasing steepness and amplitude of the systolic upstroke and the changing location of the dicrotic notch. Reprinted from Gornon,\textsuperscript{1} with permission.
appears unnaturally smooth, with a diminished or absent dicrotic notch. Overdamping results in falsely low systolic and falsely high diastolic pressure readings26 (Figures 6 and 7). An overdamped-appearing (often simply called damped) waveform can also be the result of aortic stenosis, vasodilation, or low cardiac output states such as cardiogenic shock, sepsis, or severe hypovolemia (Figure 5). In order to determine if the waveform is a result of an overdamped system or is an accurate reflection of a patient’s status, the dynamic response characteristics must be tested.

Today’s commercially available catheter-transducer systems tend to have a low natural frequency and consequently are often underdamped.22,26 Anecdotal reports of manufacturers of an improved natural frequency often do not hold up in clinical practice. Typically, the natural frequency of hemodynamic monitoring systems currently on the market is less than 25 Hz. An underdamped system will record falsely high systolic pressures (15-30 mm Hg) and falsely low diastolic pressures26 (Figures 8-10). Underdamping, most often in the form of systolic overshoot (the artificial exaggeration of systolic pressure), must be suspected in patients with hypertension, atherosclerosis, vasoconstriction, aortic regurgitation, or hyperdynamic states such as fever.23 In these conditions, typically a rapid rise in the systolic slope occurs, frequently exceeding the dynamic response characteristics of the monitoring system. A heart rate greater than 150/min may also cause systolic overshoot, because of the rapid succession of impulses.26

The variable natural frequency of available monitoring systems explains why with different monitoring systems, different hemodynamic pressures can be measured even though the patient’s condition has not changed.21 The often observed discrepancies between directly and indirectly obtained SBP and DBP readings are also partly caused by underdamping.22

Clinically, recognition of possibly overdamped and underdamped waveforms and an awareness of physiological situations when each type of waveform may occur are important. Then the question to be answered is whether the cause of the waveform is the patient’s condition or the monitoring system. Practically, the intervention for both overdamped and underdamped systems is the same: maximizing natural frequency.

Techniques for Maximizing Natural Frequency

As mentioned earlier, air bubbles are a main factor in waveform blunting or overdamping. In addition to air bubbles, other factors may alter the natural frequency of a monitoring system and distort the recorded signal. These factors include narrow, compliant, and long tubing; presence of additional stopcocks; and loose connections. Translated into practice, the monitoring system
should (1) be primed air-free, (2) consist of wide-bore, high-pressure tubing with its length limited to 122 cm (48 in), (3) not be extended with tubing or added stopcocks, and (4) have tightly secured connections. In addition, the continuous flush bag should be cleared of any air and be maintained adequately filled, and the external pressure cuff surrounding the flush solution bag should be maintained at a pressure of 300 mm Hg. This practice will not only prevent air from going into the solution but also help prevent catheter clotting.

Catheter clotting is a rare, but possibly serious, complication of intra-arterial monitoring. One of the first indications of clotting may be a waveform that looks overdamped. Whenever waveform overdamping is observed, the patient should be assessed for signs and symptoms of hypotension or low cardiac output. Then, the potential clot should be removed by aspirating blood from the distal stopcock before performing any flushing maneuver for the dynamic response test. Fluids with viscosities higher than the viscosity of normal isotonic sodium chloride solution also lead to overdamping of the hemodynamic waveform. For example, blood in the arterial catheter (due to blood backup or insufficient flushing) should be cleared from the system. As a general rule, the catheter-tubing system and stopcocks should be flushed before any hemodynamic measurement is performed, especially when clinical decisions are to be made. The Table summarizes the most important recommendations for optimizing the natural frequency of the monitoring system and dynamic response testing.

In general, overdamping and underdamping affect mostly SBP and DBP. MAP is less sensitive to these sources of waveform distortion and is therefore less dependent on the dynamic response characteristics of the catheter system. When all steps have been taken to maximize the natural frequency of a system, yet the dynamic response test indicates overdamping or underdamping, then either MAP should be followed or an alternative method of monitoring (eg, oscillometric blood pressure monitoring) should be used.

### End-Hole Artifact

The arterial catheter of the ABP monitoring system points upstream. The forward-flowing blood contains kinetic energy. When the flowing blood is suddenly stopped by the tip of the catheter, the kinetic energy of the blood is partially converted into pressure. This converted pressure may add 2 to 10 mm Hg to the systolic pressure measured by an intra-arterial monitoring system. The artificial augmentation of directly monitored systolic pres-sure by AACN on October 23, 2017...
sure by converted kinetic energy is referred to as the end-hole artifact or the end-pressure product.

**Movement Artifact**

Motion of the tubing system enhances the fluid oscillations of the system. Although the clinical significance of movement artifact is not known, it is recommended that extrinsic movement of the tubing system be kept at an absolute minimum.\(^{10}\)

**Monitor Artifact**

The differences (>5 mm Hg) between the digital pressure output displayed on the monitor and pressures directly read from analog or strip chart recordings are potentially important.\(^{43}\) These differences can lead to erroneous data collection and are especially notable in patients with hypotension, hypertension, dysrhythmias, or pulsus paradoxus.\(^{45}\) The monitoring system cannot discriminate between pressure readings during zeroing, obtaining blood samples, and fast-flushing and real arterial pressure readings. Consequently, all readings are incorporated into pressure trends.\(^{46}\) Monitoring artifact (ie, monitoring noise caused by movement of the patient and disturbances in a monitoring system due to, for example, electrical heating or cooling blankets) may also be superimposed on the patient’s pressure waveform. Experienced clinicians can recognize and eliminate some of these error sources and use a representative set of waveform tracings on a calibrated strip-chart recording to obtain the most valid assessment of a patient’s hemodynamic status.\(^{47}\) Thus, the optimal method for hemodynamic pressure measurement is to use the analog strip-chart recording.\(^{41,44}\)

**Respiratory Variation**

Normal breathing leads to changes in intrathoracic pressure, which affect cardiac output and systemic pressure. With spontaneous inspiration, intrathoracic pressure decreases. The decreased pressure is recognizable on the ABP tracing as a downward displacement of the waveform baseline. Concurrently, during inspiration, venous return to the right side of the heart is increased, augmenting right ventricular stroke volume. The increase in right ventricular stroke volume is offset by increased pulmonary vascular compliance and blood pooling during inspiratory thoracic expansion. Consequently, left ventricular stroke volume is decreased. Heart rate and SVR both increase as a compensatory reflex. The net result of this chain of reflexes is the phenomenon known as physiological pulsus paradoxus (a decrease in ABP of usually <10 mm Hg during spontaneous, unassisted inspiration).\(^{48}\) An inspiratory decrease in ABP greater than 10 mm Hg (pulsus paradoxus) may indicate cardiac tamponade or restrictive pericarditis. Pulsus paradoxus also occurs in patients with obstructive lung disease, pulmonary embolism, and severe heart failure and can be induced by mechanical ventilation.\(^{46,47}\)

During positive-pressure ventilation, the inspiratory increase in intrathoracic pressure can be recognized in the upward displacement of the baseline of the arterial pressure waveform. If a patient receiving mechanical ventilation is hypovolemic, the increase in...
intrathoracic pressure may lead to artificial augmentation of directly monitored SBP.

Current monitoring systems determine the mean of pressure readings at predetermined intervals. Respiratory artifact could lead to erroneous digital output data. The unpredictable effect of respiratory variation on arterial pressure provides a strong argument for reading and recording arterial pressure, like any other hemodynamic index, at the end of expiration by using a freeze-frame picture or, best, a strip-chart recording.

Hypertension and Atherosclerosis

Hypertension is due to age-related arterial stiffening, atherosclerotic narrowing, or renin-related vasoconstriction, all of which increase the magnitude of reflected waves. In these physiological conditions, reflected waves fuse with the systolic upstroke, resulting in a high pulse pressure and late high systolic peak, often manifested as a narrow systolic peak in the peripheral ABP waveform tracing. In addition, the diastolic wave may be reduced or disappear. Figures 4 and 11 show peripheral ABP tracings typical in patients with hypertension or atherosclerosis. In each instance, the small and narrow tip of the waveform may be an overestimation of systolic pressure and thereby central aortic pressure. As explained earlier, hypertension and atherosclerosis place high demands on the monitoring system. The arterial waveforms shown in Figures 4 and 11 could also be the result of systolic overshoot due to inadequate dynamic response characteristics of the arterial pressure monitoring system.

### Recommendations for optimizing the natural frequency of the arterial pressure monitoring system and dynamic response testing

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<th>Feature</th>
<th>Recommendations</th>
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<td><strong>Natural frequency of the monitoring system</strong></td>
<td>System requirements&lt;br&gt;Use wide-bore, high-pressure tubing no longer than 122 cm (48 in)&lt;br&gt;Avoid tubing extensions and minimize stopcocks&lt;br&gt;Ensure that all connections are tightened&lt;br&gt;Eliminate air from the flush fluid and air bubbles from the tubing system&lt;br&gt;Keep continuous flush bag filled and keep external pressure cuff at 300 mm Hg pressure&lt;br&gt;Clear access catheter and tubing system of any fluid other than isotonic sodium chloride solution&lt;br&gt;Prevention of catheter clotting&lt;br&gt;Maintain continuous flush device as described&lt;br&gt;Use heparinized flush solution&lt;br&gt;Prevention of catheter kinking&lt;br&gt;Keep cannulated extremity in a neutral or slightly extended position</td>
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<td><strong>Dynamic response test</strong></td>
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Implementation of test<br>Whenever the waveform seems overdamped or underdamped<br>Whenever physiological changes of the patient (increased heart rate, vasoconstriction) place higher demands on the monitoring system<br>After opening the system<br>Before implementing interventions or changes of interventions<br>Whenever the accuracy of the arterial blood pressure measurement is in doubt<br>At least every 8-12 hours

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Figure 11 Example of a waveform common in patients with hypertension (arterial blood pressure, 150/45 mm Hg). Note steep systolic upstroke, narrow systolic peak, diminished diastolic run-off wave, and relative decrease in the diastolic proportion of the waveform due to a heart rate of 100/min. The waveform appears underdamped.
Monitoring system. The result of the dynamic response test provides reassurance that the recorded ABP tracing is correct.

MAP is measured as the area under the pressure curve divided by the width of the base of the pressure curve (the time interval of a single cardiac cycle) (Figure 2). A small and narrow systolic tip such as the ones in Figures 4 and 11 adds relatively little to the total area under the pressure curve, whereas it can add significantly to measured SBP. Consequently, the measured MAP is less affected by wave reflection and the response characteristics of the monitoring system than is measured SBP. The MAP remains relatively constant when measured at different sites throughout the arterial circuit, whereas measured SBP and DBP may differ. In general, MAP is a more stable hemodynamic parameter and provides a more accurate interpretation of a patient’s hemodynamic status.10,34,36,38

**INDIRECT VERSUS DIRECT ABP MONITORING**

A discussion of the various methods of indirect ABP measurement and how they compare with direct ABP monitoring is beyond the scope of this article. However, several key principles should be emphasized in this context. Direct ABP monitoring measures pressure pulse, whereas all indirect methods of ABP measurement are related to blood flow. No absolute relationship exists between these 2 phenomena because they follow different laws of physics and physiology. In low-flow conditions, such as hypotension and vasoconstricted states, indirect methods yield lower pressure readings than does direct ABP monitoring. Conversely, if SVR is low, in patients with sepsis for instance, the relatively high flow results in an indirect ABP reading that is higher than the directly measured ABP. Factors such as underdamping, end-hole artifact, and wave reflection contribute to direct systolic pressure readings that are often higher than indirectly obtained pressure values. In other words, when obtaining ABP readings by using different measuring methods, different results should be expected. More specifically, a “good” correlation between the oscilloscope and the arterial pressure monitoring system is not a gauge for the proper functioning of the pressure monitoring system. MAP, on the contrary, is closely approximated when oscillometric and direct measurements are compared.21-25,36

Although both the oscillometric and the direct ABP monitoring methods are generally accurate in clinical practice, direct ABP monitoring has a distinct advantage. Direct ABP monitoring is the only scientifically and clinically validated method that allows real-time and continuous monitoring of a patient’s ABP. With strip-chart recording, beat-to-beat analysis of a patient’s ABP is possible. This feature may be of clinical relevance when evaluating the effect of positive end-expiratory pressure during mechanical ventilation or the effect of changing stroke volume in atrial fibrillation or other arrhythmias.

**SUMMARY**

Hemodynamic monitoring is a costly procedure, both materially and with regard to nursing time involved to ensure proper functioning of the monitoring system and correct interpretation of the data obtained. Dynamic response testing is the ideal method of confirming the ability of a monitoring system to accurately reproduce hemodynamic waveforms. MAP is a stable hemodynamic parameter, because it is least affected by monitoring method, catheter insertion site, the dynamic response characteristics of the catheter system, and wave reflection. MAP provides the best estimate of central aortic pressure and is the main hemodynamic parameter monitored by the neurohormonal system to control blood pressure. The superior informational value of MAP provides strong support for its preferred use in clinical practice, especially when use of vasoactive drugs is started or the dosages of these drugs are titrated.10,21 However, numerically satisfactory ABP or MAP values are not necessarily related to adequate peripheral tissue perfusion and organ system function. For optimal management of patients, data obtained from assessment tools such as hemodynamic monitoring devices must be integrated with information gained from clinical assessment of patients’ status.

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**References**

Monitoring Arterial Blood Pressure: What You May Not Know
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