9

Direct systemic arterial blood pressure monitoring

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Arterial blood pressure (ABP) measurement is one of the major hemodynamic monitoring tools used in patient assessment because adequate systemic blood pressure is required to perfuse vital organs. Arterial blood pressure, or more specifically mean arterial blood pressure (MAP), is a function of cardiac output (CO) and systemic vascular resistance (SVR). This relationship is represented by the following equation:

$$MAP = CO \times SVR \quad (9.1)$$

While ABP is often measured to assess whether systemic blood pressure is adequate to perfuse vital organs, as Equation 9.1 indicates, a normal blood pressure value does not guarantee adequate blood flow, as MAP is affected by vascular tone. The body's compensatory response to homeostatic insult, largely mediated by the sympathetic nervous system, results in tachycardia and vasoconstriction and serves to sustain blood pressure at all costs. Increasing SVR through vasoconstriction can actually diminish flow to peripheral tissues, even when ABP is maintained. Therefore, just because the blood pressure is normal does not mean blood flow is normal or that tissue perfusion is adequate. Hypotension occurs only when sympathetic compensatory mechanisms have failed after an insult.

Other monitoring techniques, such as serial physical examination, the determination of cardiac output, assessment of blood lactate concentration or central venous hemoglobin saturation, or even direct imaging of the microcirculation, potentially offer greater insight into blood flow and tissue perfusion. However, with the exception of serial physical examination and blood lactate concentration, these techniques are of limited availability and largely impractical for most practitioners. Therefore, despite the limitations of the information provided by its measurement, arterial blood pressure is still used commonly to assess hemodynamic stability in veterinary medicine. As the level of care provided to veterinary patients continues to grow, especially in a critical care setting, the value and availability of direct arterial blood pressure (dABP) monitoring has increased significantly. This chapter explores the practical and technical aspects of dABP measurement in veterinary practice.

Indications for direct arterial pressure monitoring

Blood pressure measurements provide insight into the cardiovascular status of a patient. In patients that are critically ill or have cardiovascular compromise, it is generally accepted that dABP monitoring is more accurate than methods used to obtain blood pressure indirectly (e.g., Doppler or oscillometric monitors).[1–5] Direct ABP measurement is considered to be the "gold standard" for blood pressure monitoring. There are numerous clinical scenarios in which accurate and continuous dABP monitoring would be beneficial (see Table 9.1).[6] The information obtained using dABP measurement can be used to help tailor administration of medications affecting blood pressure (e.g., titration of vasopressors or antihypertensive medications) or to help guide fluid therapy (e.g., resuscitation of hypovolemic shock) on a

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Table 9.1 Clinical scenarios benefitting from dABP monitoring[6]

- Patients in shock with hypotension or cardiovascular collapse
- Patients requiring the use of vasopressors
- Titration of medications for afterload reduction in patients with severe congestive heart failure
- Patients receiving pharmacotherapy for severe hypertension
- Patients placed on mechanical ventilation
- Patients with high anesthetic risk

minute-to-minute basis.[6] To further understand why dABP measurement might be chosen over indirect blood pressure measurement methods, it is important to understand the benefits and limitations of each modality.

Advantages and disadvantages of indirect blood pressure monitoring

As indirect arterial blood pressure (iABP) monitoring is covered in depth in Chapter 10, Noninvasive Arterial Blood Pressure Monitoring, here it is only briefly discussed in the context of comparison with dABP monitoring. Indirect methods (such as Doppler ultrasound or oscillometry) are noninvasive to the patient and therefore do not require arterial catheterization or seting up a fluid-filled monitoring system. As such, indirect methods are generally less technically demanding. While there is cost associated with acquiring equipment for iABP monitoring, it is typically much less expensive than the equipment required for dABP measurement (e.g., pressure transducers, special hemodynamic monitors). For these reasons, iABP is measured much more commonly than dAPB in both human and veterinary medicine.

Perhaps the greatest limitation to the use of iABP monitoring is its accuracy. Direct ABP measurement has been shown to be more accurate in both dogs and cats, whether awake or anesthetized.[1–5] This appears to be especially true in hypotensive, hypothermic, and small patients.[7] There are factors such as cuff size, differences in technique, and the possibility of operator error that can further affect the reliability of iABP determination. In addition, iABP measurement provides less information than does dABP measurement. For example, Doppler ultrasound technique only measures systolic blood pressure whereas the direct method measures systolic, diastolic, and mean arterial pressures. While oscillometric machines provide all three pressures, standard oscillometry may be less reliable in cats and small dogs compared with dABP or Doppler ultrasound measurement techniques.[8–9]

Advantages and disadvantages of direct arterial pressure monitoring

The dABP measurement technique offers the benefit of beat-to-beat blood pressure monitoring. This allows clinicians and technicians to monitor trends in both blood pressure and arterial waveform, thus permitting rapid recognition of changes in status and more immediate intervention. Having blood pressure readings continuously available also allows the technician to perform other treatments and monitoring, rather than spending the time necessary to acquire frequent iABP measurements. The "hands-off" nature of dABP monitoring, once it is established, may also diminish the effect that patient handling can have on the arterial blood pressure values obtained. As the stress secondary to handling may iatrogenically elevate blood pressure, dABP could allow for more accurate assessment. In addition to its role in hemodynamic monitoring, placement and maintenance of an arterial catheter also allows for ease of arterial blood sampling to monitor acid–base status and blood-gas parameters, which are typically also of great interest in the critical care patient. For more information on arterial catheterization and sampling, please see Chapter 5, Arterial Puncture and Catheterization.

Though the technique may be more accurate in critically ill patients, dABP monitoring is not without drawbacks, risks, and complications. Obtaining and maintaining arterial access can be technically challenging. Further, the equipment necessary to monitor dABP, especially continuously, can be expensive (pressure transducers, hemodynamic monitors, etc.) compared with the techniques used for indirect determination. While it is considered to be the most accurate method for blood pressure determination, there are numerous factors (both technical and mechanical) that can interfere with blood pressure signal transduction and overall accuracy of the readings, making even this gold standard prone to error. Technical issues that contribute to inaccuracy (overdamping, underdamping, zeroing errors, etc.) are discussed in detail in Chapter 8, Fluid-Filled Hemodynamic Monitoring Systems. Potential complications associated with arterial catheterization include hematoma or bleeding at the catheter insertion site, infection, arterial thrombosis and associated tissue ischemia, and significant hemorrhage if the transducer system becomes disconnected.

Continuous dABP equipment and setup

Once the decision is made to measure dABP, all of the necessary equipment must be available (see Protocols 8.1–8.4).

The first step in establishing dABP monitoring is obtaining arterial access. Arterial catheter placement is discussed in depth in Chapter 5, Arterial Puncture and Catheterization; what follows here is a brief overview. Placement of an arterial catheter can be done percutaneously or by a cutdown method. The most common arteries used for dABP monitoring in small animals are the dorsal pedal and femoral, though coccygeal can also be used. It has been demonstrated in human patients that there is no significant difference in the accuracy of pressures obtained from a peripheral as opposed to a central arterial catheter, especially with regard to MAP.[10] Patient size often plays a role in catheter insertion site as dorsal pedal access can be very challenging in cats and very small dogs. Femoral or coccygeal arteries may be better options in these patients. The catheter site should be clipped and aseptically prepared. Local anesthetics such as 2% lidocaine can be injected locally prior to the procedure to decrease patient discomfort, especially if a cutdown is performed. Special arterial catheters are commercially available; they are generally more rigid, may contain a guide wire to facilitate placement, and are intended for longer-term use. These catheters are more typically used for femoral arterial access. Most commonly an over-the-needle peripheral intravenous (IV) catheter is used. Once the area has been prepared, the artery is palpated and the catheter advanced through the skin toward the pulse. Given the relatively small lumen of arteries compared to veins, it is important that only very small incremental advances are made until there is a flash of blood in the catheter hub. Once this occurs the catheter is advanced off the stylet and into the artery. The catheter is secured with tape and appropriate protective wrap, keeping the insertion site clean and dry. It is further important to label the catheter as "Arterial Line" so that intravenous injections are not inadvertently administered. The site should be inspected daily to ensure there are no signs of bleeding or infection. Warmth of the extremity distal to the insertion site should be assessed regularly to monitor for arterial thrombosis.

Once arterial access has been established, the pressure transducer and monitoring system can be attached to the catheter (see Protocols 8.1–8.4). At one end the pressure transducer is attached via an administration set to a pressurized 500-mL or 1.0-L bag of 0.9% NaCl to which heparin has been added (to achieve a 1–2 U/mL concentration). The pressure bag must be inflated to a pressure greater than the patient's systolic blood pressure or blood will flow back into the line. Typically a pressure between 250 and 300 mm Hg is adequate unless the patient has significant hypertension. Heparinized saline is flushed through the system to prime the tubing,

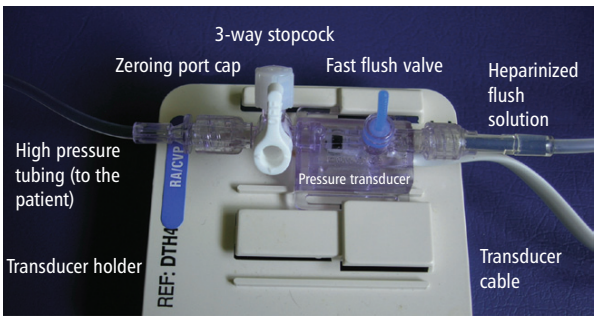

Figure 9.1 Pressure transducer for a fluid-filled hemodynamic monitoring system.

making sure to evacuate any air bubbles. When connected to the pressurized saline bag, the transducer will allow a slow forward flow of fluid through the system to decrease the risk of clot formation and catheter occlusion (check the manufacturer's materials for exact flow rates through a given transducer). Most pressure transducers also have a unidirectional flush valve ("fast flush valve") that can be used to prime the noncompliant tubing (see Fig. 9.1). At the other end of the transducer is rigid, noncompliant ("high-pressure") tubing that will be attached to the arterial catheter (see Fig. 9.1). If your transducer system does not come equipped with noncompliant tubing, you will need to supply your own and use it to complete the circuit. *It is important that standard extension tubing is not used for this purpose, as its compliant nature will result in signal distortion and affect the accuracy of blood pressure readings* (see Abnormal Arterial Pressure Waveforms below). Once the tubing is connected to the patient, the catheter is flushed to verify patency. The use of Luer lock adapters (such as Luer-Lok from Becton, Dickinson and Company, Franklin Lakes, NJ) throughout the system aids in safety and integrity. Finally, the pressure transducer is connected to the hemodynamic monitor via a transducer cable.

Before you can begin monitoring patient blood pressure you must zero the transducer. This sets a reference point (called a zero point) with which the pressure readings from the system are compared. To zero the system, the transducer should be placed at the level of the right atrium (RA) to best approximate central venous pressure. If peripheral pressures are preferred, the transducer should be placed at the level of the catheter. Once the transducer is positioned, the stopcock is closed to the patient and opened to the atmosphere, and the "zero transducer" or similar button on the monitor is engaged. The waveform line should flatten and the screen should read "0/0 (0)." When zeroing is complete the stopcock is closed to the atmosphere and opened to the patient and

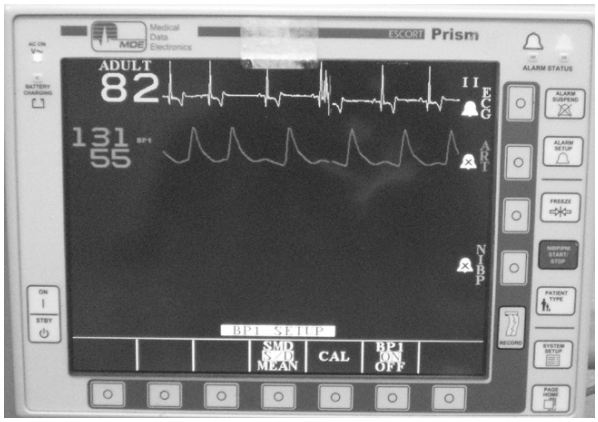

Figure 9.2 Hemodynamic monitor screen capture demonstrating standard output of arterial waveform with systolic and diastolic pressures displayed.

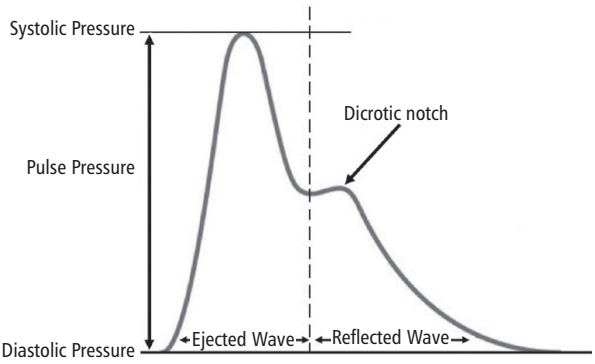

Figure 9.3 Idealized arterial pressure waveform.

the arterial waveform should appear on the screen, providing continuous arterial blood pressure measurements (see Fig. 9.2).

Normal arterial pressure waveforms

The waveform generated by the hemodynamic monitor is a reflection of the pressure changes transmitted along the arterial tree and sensed by the transducer. An idealized schematic of an arterial pressure waveform is depicted in Figure 9.3. The "baseline" of the waveform represents **diastolic arterial pressure** (DAP) and indicates the minimum blood pressure, which is present during ventricular relaxation (diastole). Diastolic arterial pressure is a function of blood viscosity, arterial distensibility, systemic vascular resistance, and the length of the cardiac cycle.[11,12] The initial upstroke in the waveform represents the rapid rise in arterial pressure from DAP to **systolic arterial pressure** (SAP), which occurs with opening of the aortic valve and stroke volume ejection. Systolic arterial pressure represents the peak blood pressure during ventricular contraction (systole), and its determinants are stroke volume, velocity of left ventricular ejection, systemic vascular resistance, arterial distensibility, and left ventricular preload.[12,13] The difference between DAP and SAP is called the **pulse pressure** and is responsible for the intensity of palpated peripheral pulses. As the stroke volume runs off into the arterial tree toward the end of systole, there is a decline in pressure (initial downslope). Once aortic pressure exceeds left ventricular pressure (with left ventricular relaxation), the aortic valve closes. Elastic recoil of the arterial tree in the presence of a closed aortic valve causes a slight rebound (or elevation) of arterial blood pressure and results in the dicrotic notch, also called the incisura (see Fig. 9.3). This notch causes disruption in the downslope of the waveform as pressures return to diastolic values. The presence of the dicrotic notch is largely a function of arterial elasticity and can be significantly diminished to absent in the face of vasoconstriction.[14]

Although the arterial waveform tends to take on the appearance just described, there are changes that occur as the pressure wave moves from central arterial circulation out to the periphery. This phenomenon is referred to as **distal pulse amplification**. As such there can be slight differences in tracings obtained depending on where the catheter tip is located. In general, the initial upstroke becomes steeper, the systolic pressure increases, the dicrotic notch appears later, and the end-diastolic pressure decreases as the waveform moves from central to peripheral (Fig. 9.4).[15] Despite the higher systolic pressure and wider pulse pressure obtained peripherally, the lower peripheral diastolic pressure results in little net effect on the mean arterial pressure from central to peripheral measurement sites.

In addition to differences in arterial pressure and waveform referable to catheter location, there can also be normal, minor variations in blood pressure seen with spontaneous as opposed to mechanical ventilation. During spontaneous breathing, SAP is slightly lower during inspiration than it is during expiration. During mechanical ventilation the opposite is true: SAP slightly increases during inspiration and decreases during expiration (Fig. 9.5). Arterial pressure changes during the respiratory cycle because alterations in pleural pressure affect thoracic vasculature and cardiac function, which in turn cause changes in stroke volume. During mechanical inspiration, positive pleural pressure results in an increase in left ventricular preload and a decrease in left ventricular afterload. The net effect is an increase in

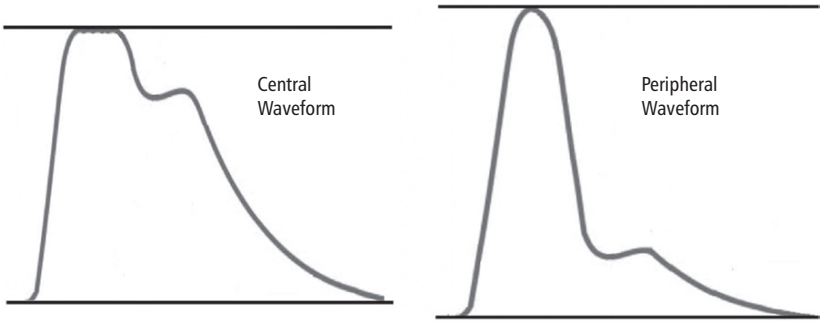

Figure 9.4 Comparison of idealized arterial waveforms from a catheter placed either centrally (left) or peripherally (right).

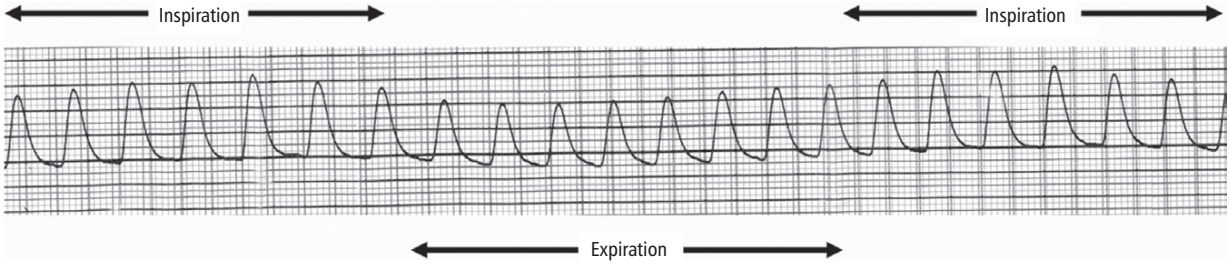

Figure 9.5 Respiratory-associated variation in arterial pressure for a patient undergoing mechanical ventilation.

left ventricular stroke volume and thereby SAP. However, pleural pressure changes also result in decreased stroke volume leading to decreased SAP during passive expiration in the mechanically ventilated patient. Under normal circumstances this pressure variation, which typically does not exceed 5 mm Hg, is not clinically significant.[16] However, as discussed later, there are certain pathological conditions that can lead to exaggeration of this respiratory cycle-related arterial pressure variation, making the variation more important both diagnostically and therapeutically.

Normal arterial blood pressures in dogs have been reported to range from 110 to 190 mm Hg for systolic and from 55 to 110 mm Hg for diastolic pressure, whereas cats have systolic pressures ranging from 120 to 170 mm Hg and diastolic pressures ranging from 70 to 120 mm Hg.[7] Mean arterial blood pressure normally ranges from 80 to 130 mm Hg in both species.

Calculations derived from the arterial pressure waveform

Mean arterial pressure is generally considered superior to systolic pressure as an indicator of true driving pressure for tissue perfusion.[17] In addition, MAP is much less susceptible to variability associated with catheter location and transducer signal distortion. As such, determination of MAP is important for assessing clinical status as well as guiding therapeutic decisions. Using dABP measurement, most hemodynamic monitors calculate MAP by averaging the area under the arterial pressure waveform over several beats. It is also possible to approximate MAP through a calculation based solely on SAP and DAP. Based on the premise that approximately two-thirds of the cardiac cycle is spent in diastole, the equation

$$MAP = DAP + (SAP - DAP)/3 \quad (9.2)$$

provides a good estimate of MAP. However, patients with tachycardia have decreased diastolic filling time, and thus this equation will underestimate MAP. In addition, this equation may overestimate MAP in patients with narrow arterial pulse pressure waveforms, as these waveforms have a smaller area under the curve (and thereby a lower true MAP), regardless of the SAP and DAP.

Calculations of arterial blood pressure variation

As previously stated, there are normally minor variations in systolic blood pressure during both spontane-

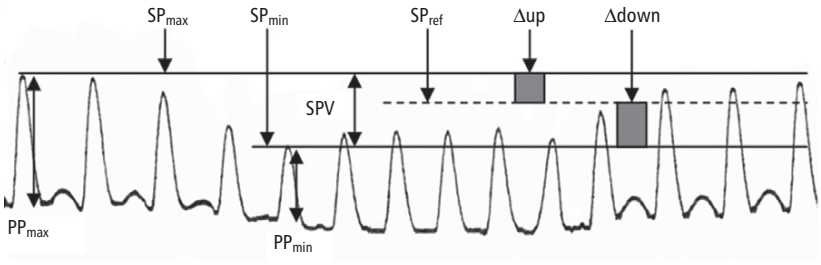

Figure 9.6 Variables used to determine volume responsiveness based on ventilation-associated variation in blood pressure: SPV, systolic pressure variation; SP_{max}, maximum systolic pressure; SP_{min}, minimum systolic pressure; SP_{ref}, reference systolic pressure; PP_{max}, maximum pulse pressure; PP_{min}, minimum pulse pressure.[16]

ous and mechanical ventilation. Hypovolemia magnifies this effect because, during hypovolemia, the heart and the thin-walled intrathoracic vessels (such as the vena cava and pulmonary veins) are more collapsible. Under such circumstances, the changes in pleural pressure that occur during the respiratory cycle can have more significant hemodynamic impact and thus result in greater pressure variation. This fact has led to the notion that respiratory cycle-associated arterial pressure variation could be used as an indicator of volume responsiveness for patients undergoing positive-pressure ventilation. Spontaneously breathing patients generally have wide variation in tidal volume, and thereby variable changes in intrathoracic pressures, which unfortunately makes respiratory effects on arterial pressure less consistent and interpretation very challenging. Systolic pressure variation (SPV), Δup/Δdown, and pulse pressure variation (PPV), all markers of respiratory cycle-associated arterial pressure variation, have been explored as indicators of volume responsiveness for patients undergoing positive-pressure ventilation and are discussed here briefly:

- *Systolic pressure variation*—Systolic pressure variation, very simply, is the difference between the maximum systolic pressure (SP_{max}) present during inspiration and the minimum systolic pressure (SP_{min}) present during expiration:

$$SPV = SP_{max} - SP_{min} \quad (9.3)$$

  An SPV greater than 10 mm Hg has been shown to correlate fairly well to hypovolemia in human patients.[18] In addition, in people, SPV has been shown to correlate with pulmonary capillary wedge pressure and left ventricular end-diastolic area, both correlates of intravascular volume status.[19,20]
- *Δup and Δdown*—As an extension of SPV, some investigators have evaluated the utility of Δup and Δdown as indicators of intravascular volume status and a patient's potential for fluid responsiveness (see Fig. 9.6). To use Δup and Δdown, one must first determine a reference systolic pressure (SP_{ref}) by measuring SAP during an end-expiratory pause. Δup is then the difference between SP_{max} and SP_{ref} and more specifically reflects the positive-pressure-inspiratory increase in SAP. Conversely, Δdown is the difference between SP_{ref} and SP_{min} and reflects the expiratory decrease in SAP:

$$\Delta up = SP_{max} - SP_{ref} \quad (9.4)$$

$$\Delta down = SP_{ref} - SP_{min} \quad (9.5)$$

  Since it is thought that the expiratory decrease in systolic pressure contributes the majority of the SPV seen with hypovolemia, it stands to reason that Δdown might be a more useful calculation than SPV. However, Δdown has not been shown to offer any better correlation to hypovolemia or volume responsiveness when compared with SPV in human patients with sepsis-induced hypotension.[21]
- *Pulse pressure variation*—Pulse pressure variation (PPV) offers yet another way to quantify respiratory variation in arterial pressures associated with ventilation (see Fig. 9.6). Pulse pressure variation is obtained by dividing the difference between the maximum and minimum pulse pressures (PP_{max} and PP_{min}) over a single breath by the mean of the two values.[22] The PPV, expressed as a percentage, is given by the following equation:

$$PPV(\%) = 100 \times (PP_{max} - PP_{min})/[(PP_{max} + PP_{min})/2] \quad (9.6)$$

  Compared with SPV, Δup, and Δdown, *PPV appears to have the strongest correlation to hypovolemia and volume responsiveness,* with higher PPVs correlating to greater degrees of volume responsiveness.[23,24]

There are several limitations to using these techniques. Perhaps the most significant is that, as previously mentioned, arterial blood pressure variation equations can only be used in patients undergoing positive-pressure ventilation. This limits the application to patients needing ventilatory support for hypoxemia, hypoventilation, or during anesthesia. In addition, factors such as technical issues, the presence of arrhythmias, effects of chest wall and lung compliance, or right or left ventricular failure could all interfere with the accuracy and utility of these values in determining volume responsiveness.[16]

Other uses for the arterial pressure waveform

In addition to use in assessment of volume status, arterial waveforms have also been used to determine cardiac output through pulse contour analysis. Pulse contour analysis provides beat-to-beat cardiac output values based on computation of the area under the systolic portion of the arterial pressure curve after calibration with a known cardiac output (typically determined by either lithium dilution or thermodilution). Cardiac output determination from the arterial pressure waveform requires additional equipment and software. Available systems include PulseCO (LiDCO Ltd, London, UK), PiCCO (Pulsion Medical Systems, Munich, Germany), and Flotrac (Edwards Lifesciences, Irvine, CA), all of which have been validated in a variety clinical scenarios in humans.[25–29] While potentially useful in clinical veterinary medicine, cardiac output determination by pulse contour analysis has been shown to have poor correlation compared with lithium dilution in patients with anesthesia or hypovolemia-induced hypotension, and frequent recalibration is required.[30,31]

Abnormal arterial pressure waveforms

Recognition of abnormal pressure waveforms is an essential component of utilizing dABP (see Protocol 9.1). Alterations in waveform morphology could reflect true changes in clinical condition, thereby warranting intervention for the patient. On the other hand, they could indicate a technical or mechanical issue that would require troubleshooting the system rather than the patient.

Technical problems that cause abnormal arterial pressure waveforms

One of the major technical issues that can arise with use of a dABP transducer system is pressure waveform overdamping or underdamping. Damping is the inherent tendency for the system itself to alter the pressure signal as it is transmitted from the patient to the transducer. Underdamping occurs when the resonant frequency of the monitoring system too closely matches the frequency of the pressure waveform. The result is a summation or resonance of the two frequencies, amplification of the signal, overestimation of SAP, and underestimation of DAP. *All dABP monitoring systems have some inherent underdamping effects and, as such, tend to report falsely high SAPs and falsely low DAPs.* The degree of inaccuracy can be minor or significant. The MAP reported is generally considered accurate. Normal arterial pressure waveforms have no "pointy" or jagged parts—waveforms with points or sharp peaks are therefore likely underdamped. The length of tubing connecting the arterial catheter to the transducer can contribute to underdamping in a direct relationship—increased length of tubing proportionally worsens underdamping.

Overdamping, on the other hand, results in attenuation or muting of the arterial pressure waveform, leading

Protocol 9.1 Suggested step-by-step approach for assessing direct arterial blood pressure (dABP)

Procedure

1. Determination of arterial blood pressure:
   a. What is the reported systolic pressure?
   b. What is the reported diastolic pressure?
   c. What is the reported or calculated mean pressure?
   d. What is the calculated pulse pressure?
   e. Do these values reflect hypotension or hypertension?
   f. Do these values match the patient's clinical condition?
   g. Do these values coincide with palpated pulse quality?
   h. Is there significant ventilation-associated variation?
2. Assessment of pulse rate and rhythm:
   a. What is the pulse rate reported by the monitor?
   b. Does this value reflect bradycardia or tachycardia?
   c. Does the rate match the auscultated and ECG heart rate?
   d. Is the pulse rhythm regular or irregular? Does it match changes in the ECG (i.e., is there a pulse waveform for each QRS complex)?
3. Assessment of waveform morphology:
   a. Has the waveform morphology changed significantly?
   b. Is the morphology consistent from beat to beat?
   c. Is a dicrotic notch present?
   d. Has the waveform become muted (significant decrease in pulse pressure)?

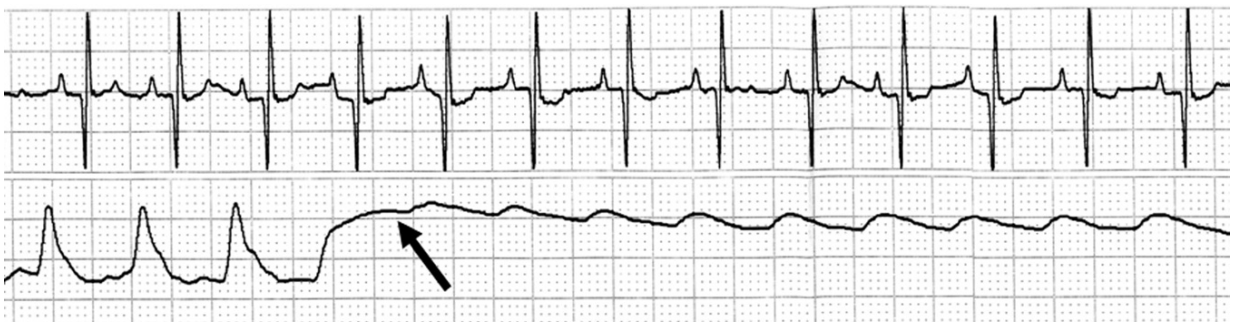

Figure 9.7 Arterial waveform from a patient with sudden and marked overdamping of the pressure signal (arrow) associated with catheter occlusion. Note the sudden loss of waveform morphology, slight decrease in systolic pressure, increase in diastolic pressure, and loss of dicrotic notch, all without any change in heart rate.

to falsely low SAP and falsely elevated DAP. The net effect is a significant reduction in the pulse pressure though the MAP generally remains relatively accurate. Overdamped waveforms are very smooth with loss of many of their defining characteristics, such as the systolic upstroke and dicrotic notch (see Fig. 9.7). Potential causes of overdamping include air bubbles in the line, line occlusion from kinking or clotting, or use of overly compliant tubing. More detail concerning system damping and technical issues of fluid-filled monitoring systems can be found in Chapter 8, Fluid-Filled Hemodynamic Monitoring Systems.

Patient problems that cause abnormal arterial pressure waveforms

Alterations in arterial waveforms can also manifest as a result of significant changes in patient hemodynamics. For example, various arrhythmias can have a significant impact on cardiac output and blood pressure, and can result in diminished to completely absent arterial waveforms despite the presence of electrical activity (see Fig. 9.8).

Significant hypotension secondary to low cardiac output (as from hypovolemic or cardiogenic shock) can result in markedly muted waveforms secondary to small stroke volumes combined with peripheral vasoconstriction. As such, pressure waveforms from patients with hypotension can be difficult to distinguish from those caused by overdamped systems. Clearly, recognizing the difference is essential to taking appropriate action if the patient is truly hypotensive. The patient's clinical status (mental responsiveness, heart rate, manual palpation of pulses) as well as the MAP (remembering that MAP is typically preserved with overdamping and will be low with hypotension) can be helpful in distinguishing between the two. Alternately, a fast flush test (described in Chapter 8, Fluid-Filled Hemodynamic Monitoring Systems) can be performed to assess the system for damping.

Another manifestation of respiratory arterial pressure variation can occur in the form of pulsus paradoxicus, most commonly associated with pericardial effusion that has resulted in cardiac tamponade. Similar to hypovolemia, the effective decrease in venous return from increased pericardial pressure results in an exaggeration of the difference between the SAP during inspiration and the SAP during expiration. Provided the patient is breathing spontaneously, SAP will be higher on expiration and lower on inspiration (see Fig. 9.9a).

Finally, there are certain clinical scenarios whereby a patient has waveforms with increased pulse pressure ("tall") but are of fairly short duration ("narrow") (see Fig. 9.9b). This morphology is typically caused by an increased SAP and a very rapid falloff to DAP, the latter occurring either because of decreased blood viscosity or backward flow of blood. Potential causes of "tall and narrow" waveforms, also referred to as water hammer or Corrigan's pulses, include aortic regurgitation, patent ductus arteriosus, hypertension, and hemodilutional anemia.[32]

Thresholds of concern for arterial pressure value and waveform abnormalities

Significant changes in blood pressure or waveform morphology should prompt assessment of clinical condition and intervention as indicated (see Table 9.2). Onset or worsening of hypotension, generally defined as SAP less than 80 mm Hg or MAP less than 60 mm Hg, should be addressed as soon as possible to limit tissue ischemia and potential for cardiac arrest.[7] Along similar lines, marked hypertension, generally defined as SAP greater

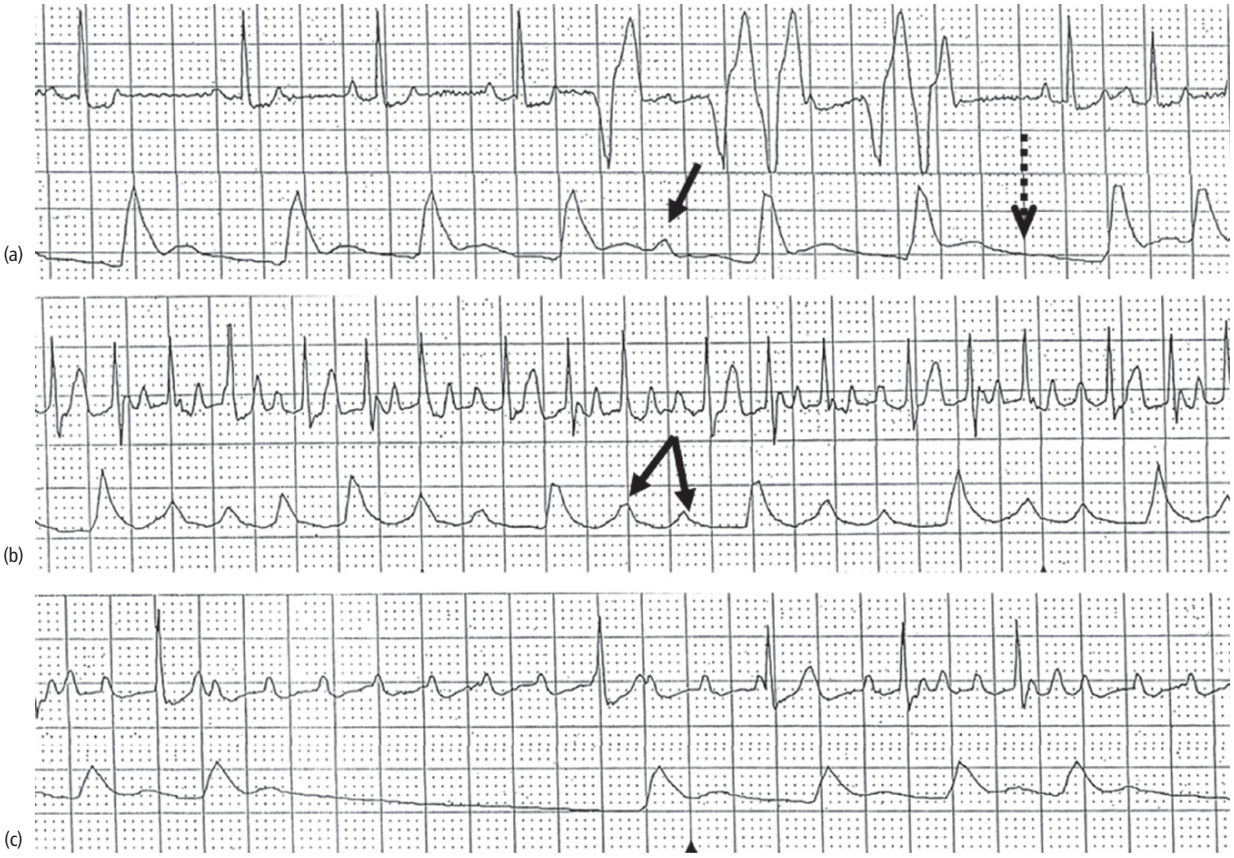

Figure 9.8 Examples of arrhythmia-associated changes in arterial waveform morphology. (A) Ventricular premature contractions associated with diminished (solid arrow) to absent (dashed arrow) pressure tracings. (B) Marked tachycardia (heart rate 210 beats per minute) resulting in progressively diminished waveforms and blood pressure (arrows). (C) Atrial flutter with prolonged periods of absent ventricular contraction resulting in absent arterial waveforms.

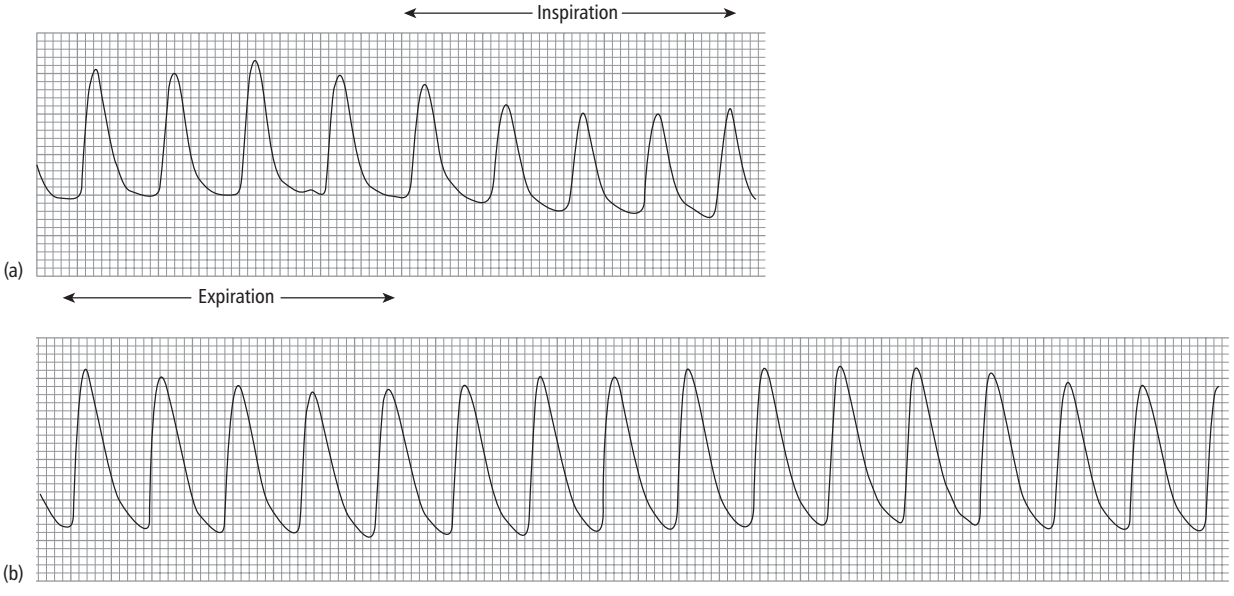

Figure 9.9 (a) Arterial waveforms from a patient with pulsus paradoxicus demonstrating respiratory-associated arterial pressure variation. (b) Arterial waveforms from a patient with hemodilutional anemia demonstrating "tall and narrow" morphology.

Table 9.2 Guidelines indicating need for clinician intervention based on dABP monitoring

- Hypotension[7]
  - SAP < 80 mm Hg
  - MAP < 60 mm Hg
- Hypertension[33]
  - SAP > 180–200 mm Hg
  - MAP > 140 mm Hg
- Arrhythmias[35]
  - Tachyarrhythmias
    - HR > 180–200 (dog)
    - HR > 240 (cat)
  - Bradyarrhythmias
    - HR < 60 (dog)
    - HR < 80–100 (cat)

than 180–200 mm Hg or MAP greater than 140 mm Hg, could cause significant end-organ injury and would require intervention.[33] The presence of ventilatory variation in systolic pressure greater then 10 mm Hg might suggest hypovolemia (prompting more aggressive fluid resuscitation) or pericardial effusion (prompting pericardiocentesis).[34] Arrhythmias that result in a sustained impact on blood pressure or even intermittent hypotension would need to be addressed with antiarrhythmic medications or a pacemaker. This could include tachycardia with heart rate greater than 180–200 beats per minute (bpm) in dogs or 240 bpm in cats, or bradycardia with heart rate less than 60 bpm in dogs and 80–100 bpm in cats.[35]

Troubleshooting abnormal waveforms

One of the primary objectives in troubleshooting abnormal waveforms is to determine whether changes in blood pressure or waveform morphology indicated by dABP monitoring are truly reflective of hemodynamic changes in the patient or if they are a function of technical or mechanical issues. These issues arise most commonly with the apparent presence of significant hypotension (and muting of the arterial waveform) or hypertension (and exaggeration of the waveform). Several steps can be followed to systematically work through the potential causes for these changes (see Protocol 9.2). When an abnormal waveform is detected, the first step is to determine if there are concurrent changes in the patient's clinical status such as alteration in responsiveness, heart rate, or palpable pulse quality that might require immediate intervention. In addition, if not already being continuously monitored, an ECG should be obtained to determine if an arrhythmia is present. If there are not any readily discernable changes in clinical condition or ECG, the transducer setup should be assessed to make sure that the noncompliant tubing is not kinked and that there are no air bubbles present in the fluid column. It is also important to ensure the pressure bag is still inflated to at least 250 mm Hg. If there has been significant change in patient position, it may be beneficial to relevel and rezero the transducer. To assess patency of the arterial catheter, the catheter should be aspirated to ensure arterial blood is obtained; any air bubbles or clots should be removed. If the catheter does not readily aspirate, the system can be gently, manually flushed to assess patency and to evacuate any air bubbles or clots. A fast or forceful flush of an arterial catheter that does not readily aspirate carries the risk of introducing air or small thrombi into distal arterial circulation. If the system does not flush or aspirate readily, the arterial catheter should be closely inspected to confirm its position and functionality. If the catheter appears to be at least partially patent then a "fast flush test" (described in detail in Chapter 8, Fluid-Filled

Protocol 9.2 Troubleshooting abnormal arterial pressure waveforms

Procedure

- Assess patient for changes in clinical status to explain change in morphology:
  - Assess mentation, heart rate, pulse quality, mucous membrane color, capillary refill time, and extremity temperature.
  - Assess ECG for changes in rate and rhythm coinciding with changes in waveform.
- If patient parameters have not changed:
  - Assess transducer setup and make sure:
    - Noncompliant tubing is used between patient and transducer.
    - Noncompliant tubing is not kinked.
    - There are no air bubbles in the fluid column.
    - Pressure bag is inflated to at least 250 mm Hg.
  - Relevel transducer to level of the patient's heart base and rezero the transducer, if patient position has changed significantly.
  - Assess patency of arterial catheter:
    - Aspirate arterial catheter.
      - Ensure arterial blood is easily obtained.
      - Remove air bubbles and clots from line.
    - Flush arterial catheter.
      - Ensure catheter flushes easily—do not force.
      - Evacuate air bubbles or small clots from system.
- Perform "fast flush test" to assess for system overdamping or underdamping.

Hemodynamic Monitoring Systems) can be performed to assess for the presence of overdamping or underdamping. If underdamping is present, it may be necessary to use shorter noncompliant tubing between the pressure transducer and the arterial catheter. If overdamping is present and does not resolve with flushing, it may be necessary to confirm the presence of noncompliant rather than standard extension tubing between the catheter and the transducer, and to assess all system connections and the arterial catheter itself.

Conclusions

Despite the potential limitations and technical difficulties associated with its use, dABP monitoring offers valuable information regarding hemodynamic status. If equipment is available and a level of familiarity is obtained, dABP measurement could be used in any 24-hour veterinary hospital and is especially useful if critically ill patients are routinely seen.

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