CHAPTER 183

HEMODYNAMIC MONITORING

Lori S. Waddell, DVM, DACVECC • Andrew J. Brown, MA, VetMB, MRCVS, DACVECC

KEY POINTS

- Hemodynamic monitoring is essential in the treatment of many critically ill patients because it is important in guiding fluid and pharmacologic therapy to optimize cardiovascular function.
- Assessment of hemodynamic status is typically based on physical examination parameters and monitoring of any of the following: continuous electrocardiogram, arterial blood pressure, central venous pressure, mixed venous and central venous oxygen saturation, pulmonary artery pressure, lactate and base deficit, and cardiac output measurements.
- Continuous electrocardiogram monitoring enables the clinician to detect intermittent arrhythmias, determine whether treatment is indicated, and monitor therapeutic effectiveness.
- Direct blood pressure monitoring is the gold standard for blood pressure measurement, but indirect blood pressure monitoring is more readily available and better tolerated by most patients.
- Central venous pressure is relatively easy to monitor and can guide fluid therapy, particularly in patients that are hypovolemic or have septic shock, heart disease, or renal disease.

Hemodynamic monitoring includes monitoring of basic physical examination parameters, continuous electrocardiogram (ECG) and blood pressure, central venous pressure (CVP), central venous oxygen saturation (ScvO$_2$), and lactate clearance and base deficit, as well as the most advanced forms including pulmonary artery pressure (PAP) monitoring, mixed venous oxygen saturation (SvO$_2$) monitoring, and other technologies to measure cardiac output, cardiac index, systemic vascular resistance, oxygen delivery, and oxygen uptake. See Chapters 184 and 202 for further information on these procedures. The type of monitoring chosen depends on the severity of illness, equipment availability, and the clinician's comfort with the various modalities.

CONTINUOUS ELECTROCARDIOGRAM MONITORING

Continuous ECG monitoring can be very useful in critically ill patients; it provides continuous, hands-off access to the heart rate and rhythm. It allows the clinician to catch arrhythmias that may be intermittent and infrequent, and monitor the need for treatment based on the rate and rhythm. Both standard and telemetric systems are available, with the telemetric models allowing for easier patient movement and less tangling and disconnection of the leads compared with standard systems.

BLOOD PRESSURE MONITORING

Arterial blood pressure monitoring is extremely useful in critical cases and is commonly employed to permit fluid therapy to be tailored to the patient's needs, especially when combined with monitoring of physical examination parameters, urine output, and CVP. It is essential in guiding the use of inotropic agents and vasopressors; these therapies should not be used unless blood pressure can and will be measured frequently. Normal arterial blood pressure values for dogs are as follows: systolic pressure, 150 ± 20 mm Hg; mean pressure, 105 ± 10 mm Hg; and diastolic pressure, 85 ± 10 mm Hg. For cats, normal ranges are 125 ± 10 mm Hg for systolic, 105 ± 10 mm Hg for mean, and 90 ± 10 mm Hg for diastolic.[1] Mean arterial blood pressure can be calculated from these measured values as follows:

$$\text{Mean arterial blood pressure} = \text{diastolic} + \frac{\text{systolic} - \text{diastolic}}{3}$$

Hypotension is defined as a systolic blood pressure of less than 90 mm Hg or a mean arterial pressure of less than 60 mm Hg in either species. Causes of hypotension include decreased cardiac output secondary to reduced circulating volume, myocardial failure, severe bradyarrhythmia or tachyarrhythmia, or decreased systemic vascular resistance due to peripheral vasodilation secondary to sepsis or systemic inflammatory response syndrome. Treatment of hypotension should always be aimed at correcting the underlying problem (see Chapter 8).

Hypertension can be primary (essential hypertension), which is rare in both cats and dogs, or secondary to another disease process that alters renal or neurohormonal function. Kidney injury or failure, whether acute or chronic, is the most frequent cause of secondary hypertension, but hyperthyroidism, diabetes mellitus, hyperadrenocorticism, pheochromocytoma, and various medications (glucocorticoids, cyclosporine A, phenylpropanolamine, and erythropoietin) have also been associated with hypertension.

Blood pressure monitoring can be divided into two main categories, noninvasive and invasive methods. The noninvasive oscillometric or Doppler methods are used most commonly in veterinary patients, although photoplethysmography is also available. Invasive blood pressure monitoring provides direct arterial pressure measurement and is the most accurate method available.

Noninvasive Blood Pressure Monitoring

Noninvasive blood pressure monitoring is based on inflation of a cuff to occlude arterial flow, followed by measurement of the pressure at which flow returns. These methods are technically easy to use and require relatively inexpensive equipment but are prone to error,

usually due to selection of an inappropriate cuff size. The guideline for the cuff width is approximately 40% of the circumference of the limb for dogs and 30% of the circumference of the limb for cats. If the cuff is too small, a falsely high pressure will be obtained; if the cuff is too large, a falsely low reading will result.[2] The cuff should be at the level of the right atrium while measurements are being obtained. Keeping the patient motionless in lateral recumbency is ideal for obtaining accurate indirect blood pressure measurements.

The Doppler method is used most commonly in smaller animals such as cats, very small dogs, and exotic species. It is also useful in patients with hypotension or those that have arrhythmias because the oscillometric methods are commonly inaccurate or do not give any readings at all in these circumstances. The Doppler method uses a 10-MHz ultrasound probe to detect blood flow in an artery. The probe is placed over an artery distal to the cuff. Doppler sounds become audible when pressure in the cuff is less than the pressure in the artery. Although the Doppler method typically is regarded as measuring the systolic pressure, one study that compared Doppler readings with direct blood pressure monitoring in anesthetized cats found that the Doppler reading consistently underestimated systolic pressures by 10 to 15 mm Hg and was more closely correlated with mean arterial pressure. This study was performed only in anesthetized healthy cats, so limitations are present.[3]

The oscillometric method of blood pressure determination is commonly used in veterinary medicine. There are a number of different veterinary systems available. The cuff is alternately inflated and deflated, and during deflation alterations in cuff pressure due to pulse pressure changes are sensed by the transducer. The peak amplitude of oscillations equals the mean arterial pressure. Systolic pressure equals the pressure at which oscillations are first detected, and diastolic pressure equals the pressure at which oscillations decrease rapidly.

Oscillometric machines calculate systolic and diastolic blood pressure from the mean arterial pressure using built-in algorithms, so that the mean arterial pressure is the most accurate value. The heart rate is measured as the number of oscillations occurring per minute and should always be compared with the patient's heart rate as determined manually or by ECG. Many of the oscillometric units have been used in studies comparing systolic, mean, and diastolic pressures in anesthetized and awake dogs and cats with variable results, some of which showed acceptable correlation between values obtained with these units and direct arterial blood pressure measurements.[4-9] Later-generation units claim to be more accurate in smaller dogs and cats, but these claims have not held up in more recent studies. Poor agreement was seen using one oscillometric unit in anesthetized dogs compared with direct arterial blood pressure measurements.[10] Readings obtained using three different units were compared with direct arterial blood pressure measurements in anesthetized cats and had poor correlation.[11] Another study showed that pressures measured in awake, ill dogs using the Doppler method and two oscillometric units also were not well correlated with values obtained via direct arterial blood pressure monitoring.[12] Although none of the units currently available would meet validation criteria for humans, these units are readily available, are simpler to use and are associated with fewer potential complications than direct arterial blood pressure monitoring. The American College of Internal Medicine recently drafted a consensus statement on hypertension and proposed new validation recommendations for veterinary devices.[1]

High-definition oscillometry (HDO) is a newer modality for blood pressure monitoring in veterinary medicine. HDO devices are purported to have advantages over standard oscillometric monitors because HDO performs real-time analysis of arterial wall oscillations to obtain pressure-wave amplitudes, so systolic and diastolic pressures are measured instead of calculated. Other reported benefits include accurate readings of values from 5 to 300 mm Hg and high-speed analysis that allows for measurements at heart rates of up to 500 beats/min and during arrhythmias. However, recent studies have not shown good correlation with other blood pressure monitoring methods, although none compared HDO with direct arterial blood pressure monitoring.[13,14] More studies are needed to evaluate HDO, including studies comparing HDO results with values obtained via direct arterial blood pressure monitoring.

Photoplethysmography

Originally designed for use on the human finger, photoplethysmography is based on the "volume clamp" principle. The blood volume in an extremity varies in a cyclic pattern with each cardiac cycle. The variation is detected by a photoplethysmograph attached to a finger (or to the foot or tail in veterinary patients). If the cuff is inflated and deflated fast enough to maintain a constant volume in the finger (or distal extremity), the cuff pressure will equal intraarterial pressure. This allows for a constant, real-time display of cuff pressure, and therefore intraarterial pressure, and measurement of systolic and diastolic pressures.[15] Photoplethysmography has been evaluated in dogs and cats and found to be accurate, but has not come into common use.[3,8]

Invasive Blood Pressure Monitoring

Invasive or direct arterial blood pressure monitoring is considered the gold standard for blood pressure measurement in both veterinary and human patients, both awake and anesthetized. It is usually performed after inserting an arterial catheter that is connected to a pressure transducer and monitor, which allows for continuous monitoring of systolic, diastolic, and mean pressures. Techniques for direct arterial puncture and single-pressure measurement have also been described. See Chapter 201 for further details on placement of these catheters.

When a display monitor is employed, continuous direct arterial pressure monitoring allows for observation of pressure changes and trends (Figure 183-1). Another advantage of placing an arterial catheter is that it can be used to obtain blood samples for arterial blood gas analysis and laboratory testing. Despite its many advantages, direct monitoring should be limited to critically ill patients that will benefit from having blood pressure measured continuously over a defined period (e.g., during anesthesia in a patient with a high anesthetic risk or while hospitalized in an intensive care unit).

Direct arterial blood pressure monitoring in patients with hypovolemic or septic shock is extremely helpful in guiding volume replacement and the use of pressors to maintain an acceptable systemic blood pressure. By evaluating the pressure waveform with various arrhythmias, the clinician can distinguish which ones are causing poor pressures or even pulse deficits, and this can influence the decision to initiate treatment. Direct arterial blood pressure monitoring is not indicated in active, relatively healthy patients because of possible morbidity from arterial catheter placement and the risk of hemorrhage due to disconnection of the arterial line or premature removal by the patient. Animals with arterial catheters must be strictly supervised at all times.

Once an arterial catheter is placed, it is connected to semirigid tubing that has been primed with heparinized saline from a bag of 0.9% sodium chloride with 1 unit of heparin per milliliter of saline. The fluid bag is pressurized to 300 mm Hg to prevent backward flow of arterial blood into the tubing.[16] The tubing from the catheter is attached to a pressure transducer that is connected to a cable and mounted on a board placed at the level of the patient's heart. The pressure transducer converts the pressure changes into an electrical signal that is carried to the monitor by the transducer cable, and then the signal is amplified and displayed on a monitor as a pressure
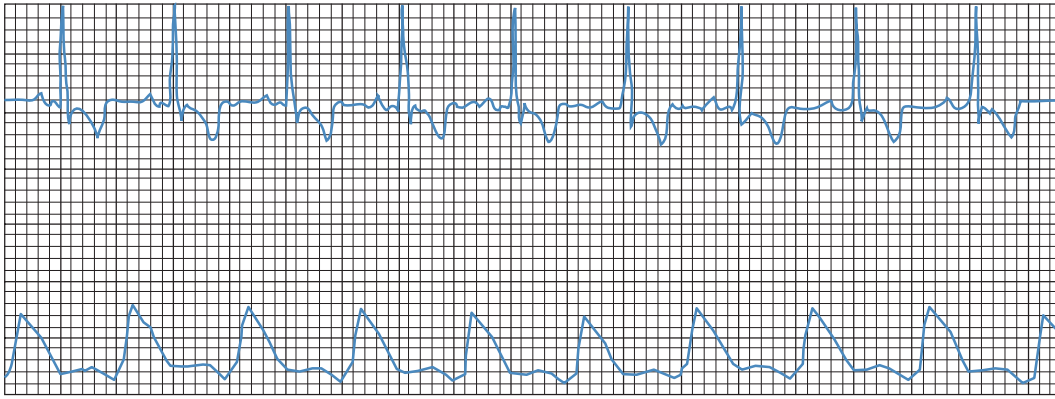
FIGURE 183-1 Direct arterial blood pressure waveform and continuous electrocardiogram. Note that one arterial pressure waveform is seen just after completion of each cardiac complex.

waveform showing the peak systolic pressure, dicrotic notch (which is created by closure of the aortic valve), and diastolic pressure. Monitors can also display numeric values for the systolic, diastolic, and mean arterial pressures.

Although direct arterial monitoring is considered the gold standard for blood pressure monitoring, it can give erroneous results if compliant tubing is used, the catheter is lodged up against the arterial wall, a clot forms at the tip of the catheter, air bubbles are present in the catheter or tubing, or the catheter or tubing becomes kinked. All of these problems can result in the waveform becoming damped, which gives falsely low systolic and falsely high diastolic values. Direct arterial blood pressure monitoring is associated with higher morbidity than noninvasive methods, including hematoma formation at the site of arterial puncture, infection, thrombosis of the artery, or necrosis of the tissues distal to the catheter (particularly in cats that have an indwelling catheter for longer than 6 to 12 hours). Keeping the arterial line patent requires heparinization of the line and catheter, which can be of concern in very small patients. Fortunately, all of the complications other than hematoma formation are quite rare.

Telemetric Blood Pressure Monitoring

Telemetric units are available for implantation into dogs and potentially cats (Data Sciences International, St. Paul, Minn.). These require surgical implantation of a transmitting device that sends digital information to a receiver; this information can then be either collected by a computer and evaluated later or converted into an analog signal for recording on a strip chart.

The device is placed subcutaneously and has a polyurethane catheter with an antithrombogenic coating and a biocompatible gel at the end that is fed into the femoral artery. This technology has been used in laboratory settings for a number of years and is currently used experimentally in both feline and canine patients. These devices allow for free patient movement and prevent the stress of handling and restraint from affecting the blood pressure measurements obtained. These devices are not used commonly in clinical patients but may be a viable option in the future for those that require long-term hospitalization or repeated blood pressure monitoring.

CENTRAL VENOUS PRESSURE MONITORING

CVP is the hydrostatic pressure in the intrathoracic vena cava and, in the absence of a vascular obstruction, is approximately equal to right atrial pressure. When the tricuspid valve is open, right atrial pressure equals right ventricular end-diastolic pressure. This pressure is used to estimate right ventricular end-diastolic volume and the

relationship between blood volume and blood volume capacity. It also gives a measure of the relative ability of the heart to pump the volume of blood that is returned to it. Patients that most commonly benefit from CVP monitoring include those that are hypovolemic or have septic shock, heart disease, or renal disease (especially oliguric or anuric kidney injury).

CVP monitoring typically requires a central venous catheter, usually a 16-gauge or 19-gauge jugular catheter, but a femoral vein catheter that extends into the abdominal vena cava has been shown to measure CVP accurately in cats without significant intraabdominal disease[17] and in puppies.[18] The size of the catheter has no effect on measurement of CVP.[19] A study evaluating the correlation between peripheral venous pressure and CVP in awake dogs and cats found that peripheral venous pressure could not be used to approximate CVP.[20]

The tip of the catheter should be positioned in the cranial or caudal vena cava just outside of the right atrium. The catheter is then connected to a three-way stopcock via noncompliant tubing and to a manometer containing heparinized saline or to a pressure transducer as described earlier for direct arterial blood pressure monitoring. The central catheter can be used for CVP monitoring as well as fluid administration or intermittent blood sampling. However, if the CVP is to be monitored continuously and the patient requires additional venous access, a multilumen venous catheter should be placed so that the other ports remain available for fluid therapy, infusions, and blood sampling. Double-lumen and triple-lumen catheters are available in a variety of sizes and lengths (see Chapter 195).

When the central venous catheter is connected to the system, the zero reference point for the bottom of the manometer or the pressure transducer should be the manubrium for a patient in lateral recumbency or the point of the shoulder for a patient in sternal recumbency. Normal ranges for mean CVP are 0 to 5 cm H_2O, but they can vary in individual animals.[21] This makes trends in the CVP much more significant than individual readings. Values can be affected by patient position, so a consistent position should be used when comparing values. Catheter position also affects readings and can be confirmed by radiography or fluoroscopy.

A recent study evaluated the use of ultrasonographically measured hepatic vein diameter, caudal vena cava diameter, and hepatic venous flow velocities, which are multiphasic and correlate with changes in the cardiac cycle in Foxhounds. The investigators found that CVP could be predicted by a multiple linear regression equation using a combination of caudal vena cava diameter, hepatic vein diameter, and the velocity of the v wave (the small retrograde wave that occurs during right atrial overfilling near the end of the T wave of

the ECG complex—see the later discussion of CVP waveform analysis).[22] This study was limited to Foxhounds that did not have a lot of variation in size; therefore it is difficult to know if this technique would be useful in other breeds or sizes of dogs.

The CVP varies throughout the respiratory and cardiac cycles because CVP reflects right atrial pressure. During inspiration, intrathoracic pressure decreases and the CVP falls. The reverse occurs during exhalation. If a patient has an upper airway obstruction and has difficulty inspiring, these changes will be exaggerated. Positive pressure ventilation will reverse this pattern.

The complexity of the CVP waveform can be seen when it is displayed on a monitor, and the variations that occur during the cardiac cycle can be observed (Figure 183-2, *A and B*). Three positive waves are seen, *a, c,* and *v* waves, and two negative depressions are seen, x and y descents. The *a* wave represents the increase in the CVP caused by right atrial contraction. The *c* wave is caused by bulging of the tricuspid valve into the right atrium, which increases right atrial pressure and CVP as the right ventricle contracts. The *x* descent is caused by the decrease in atrial pressure during ventricular ejection. The *v* wave is caused by increasing pressure from blood flowing into the right atrium before the tricuspid valve opens. The *y* descent represents rapid emptying of the right atrium as the tricuspid valve opens, allowing blood to flow into the right ventricle. Careful
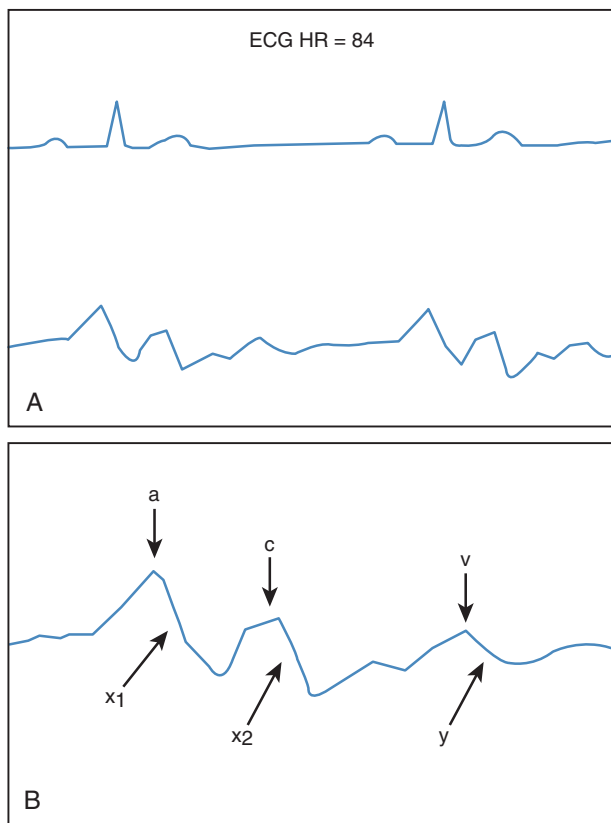

FIGURE 183-2 **A,** Central venous pressure (CVP) waveform and continuous electrocardiogram. Each phase of the cardiac cycle is reflected in the CVP waveform. **B,** CVP waveform with waves and depressions labeled. *a, a* wave, which represents the increase in the CVP caused by right atrial contraction; *c, c* wave, caused by bulging of the tricuspid valve into the right atrium; *v, v* wave, caused by increasing pressure from blood flowing into the right atrium before the tricuspid valve opens; x_1, x_1 descent; x_2, x_2 descent, caused by decreased atrial pressure during ventricular ejection; *y, y* descent, which represents rapid emptying of the right atrium as the tricuspid valve opens.

evaluation of the waveform allows abnormalities in each part of the cycle to be detected and differential diagnoses to be considered; for example, large *c* waves are often associated with tricuspid regurgitation.[23,24]

A low CVP (<0 cm H_2O) indicates hypovolemia due to fluid loss or vasodilation secondary to decreased peripheral venous resistance. A high CVP (>10 cm H_2O) may indicate volume overload, right-sided heart failure, or significant pleural effusion.[25,26] CVP readings of higher than 16 cm H_2O often lead to edema formation or body cavity effusions. Some causes of right-sided cardiac dysfunction are right-sided myocardial failure, pericardial effusion and tamponade, restrictive pericarditis, and volume overload from excessive intravenous fluid administration.

If a CVP reading is questionable, a small test bolus of 10 to 15 ml/kg of an isotonic crystalloid or 5 ml/kg of a synthetic colloid may be given over 5 minutes or less (see Chapter 58). The vascular bed is a very compliant system, able to accommodate changes in volume with minimal changes in pressure. If the patient has a low CVP due to hypovolemia, the CVP will either show no change or will have a transient rise toward normal, then rapidly decrease again. The mean arterial pressure may also increase with the test bolus, then return toward prebolus measurements. A small increase of 2 to 4 cm H_2O with a return to baseline within 15 minutes is usually seen in euvolemic patients. A large increase (>4 cm H_2O) and slow return to baseline (>30 minutes) is seen in hypervolemic animals or those with reduced cardiac compliance.[26]

Contraindications for CVP measurement are few and relate to central venous catheter placement. These include coagulopathies that would make puncture of the jugular or femoral vein an unacceptable risk; high risk of thromboembolic disease, such as in animals with protein-losing nephropathy, hyperadrenocorticism, or immune-mediated disease; and suspicion of increased intracranial pressure, such as in patients with head trauma, seizures, or intracranial disease.

The biggest limitation of CVP monitoring is that it measures the pressures on the right side of the heart instead of the left side because it is the left side that supplies the systemic circulation and drains the pulmonary circulation. Pressures in the left side are more accurate in guiding fluid therapy, but their measurement requires use of a pulmonary artery catheter, which is much more expensive, risky, time consuming, and technically challenging. This makes CVP more readily available and acceptable as an alternative to PAP and pulmonary artery occlusion pressure (PAOP) monitoring.

PULMONARY ARTERY PRESSURE MONITORING

PAP monitoring requires that a catheter be placed in the jugular vein, through the right atrium and ventricle, and into the pulmonary artery. A pulmonary artery catheter allows for measurement of the systolic, diastolic, and mean PAP (see Chapter 202). If the catheter is equipped with a balloon, PAOP (also called the *pulmonary wedge pressure*) can be measured when the balloon at the end of the catheter is inflated in a distal branch of the pulmonary artery. Inflation of the balloon eliminates PAP created by blood flow, and the measured pressure reflects the left atrial filling pressure as it equilibrates across the pulmonary capillary bed.

When the mitral valve is open, left atrial pressure equals left ventricular end-diastolic pressure. This pressure provides the best measure of left ventricular preload and is the best predictor of pulmonary edema secondary to fluid overload. Preload is the amount of stretch in the ventricle at the end of diastole and is an important determinant of cardiac output.

Like CVP, PAP and PAOP can be used for (and are more accurate at) determining the fluid volume status of a patient. Normal PAOP in dogs is 5 to 12 mm Hg.[23] Low PAOP usually signals volume

depletion and the need for fluid administration, whereas increased PAOP is indicative of volume overload or cardiac dysfunction so that additional fluid is contraindicated.

Additional parameters that can be monitored with a Swan-Ganz type of catheter are right atrial pressures (used in place of CVP), which are measured via the proximal port of the catheter, and cardiac output, which is determined using the thermodilution technique (thermodilution cardiac output). A known quantity of solution (either saline or 5% dextrose) at a known temperature is injected rapidly into the proximal port of the catheter. The cooler solution mixes and cools the surrounding blood, and the temperature difference is sensed by a thermistor at the distal tip of the catheter. The change in temperature is plotted on a time-temperature curve. The area under the curve is inversely proportional to the cardiac output, which is calculated by a cardiac output monitor. Normal values for cardiac output are 125 to 200 ml/kg/min for dogs and 120 ml/kg/min for cats.[27,28]

Other values that can be calculated include cardiac index (cardiac output ÷ body surface area in square meters), stroke volume (cardiac output ÷ heart rate), stroke volume index (stroke volume ÷ body surface area), systemic vascular resistance ([mean arterial pressure − right atrial pressure] ÷ cardiac index), and pulmonary vascular resistance ([mean PAP − PAOP] ÷ cardiac index).[28] Some catheters are also equipped with an oximeter that will measure central venous hemoglobin saturation (SvO_2). This information, combined with the arterial oxygen saturation (SaO_2), allows for determination of the oxygen content of both arterial and mixed venous blood, oxygen delivery, oxygen consumption, and oxygen extraction (see Chapters 184, 186, and 202).

Placement of these catheters is not without risk because arrhythmias, damage to the tricuspid and pulmonic valves, rupture of a pulmonary artery, and pulmonary thromboembolism have all been reported in humans undergoing the procedure.[29]

MIXED VENOUS AND CENTRAL VENOUS OXYGEN SATURATION

Measurement of SvO_2 (mixed venous oxygen saturation) and $ScvO_2$ (central venous oxygen saturation) is also useful for cardiovascular monitoring. SvO_2 is measured from the pulmonary artery, as mentioned earlier in the section on PAP monitoring, and therefore requires the placement of a catheter in the pulmonary artery. $ScvO_2$, which can be measured from a catheter in the vena cava or the right atrium, is much more accessible. Samples of blood can be removed via these catheters and analyzed with a co-oximeter or a fiberoptic fiber can be embedded in a centrally placed catheter and attached to a monitor for real-time measurements. The normal SvO_2 is greater than 75% and $ScvO_2$ is normally greater than 65%. Typically there is a very strong correlation between the two values, although they can differ by up to 18% in severe shock states.[30,31]

Tissue hypoxia causes increased extraction of oxygen from venous blood, which results in a decrease in both SvO_2 and $ScvO_2$. Increased venous oxygen extraction and resulting venous desaturation is one of the major compensatory responses to help maintain delivery of oxygen to the peripheral tissues in low flow states. Measurements of SvO_2 and $ScvO_2$ reflect systemic oxygen balance and cumulative oxygen debt.

The importance of measurement and optimization of $ScvO_2$ was highlighted in the Rivers et al study in 2001.[32] In this study, patients with severe sepsis or septic shock were treated according to an early goal-directed therapy (EGDT) protocol. One of the endpoints of resuscitation was a $ScvO_2$ of greater than 70%. The goals were to be met within the first 6 hours of therapy. The $ScvO_2$ was increased through the use of vasoactive agents, red blood cell transfusions, and

inotropes, in addition to standard therapy. The EGDT group had a significantly lower mortality rate than the conventionally treated group.[32] The EGDT group also had reduced organ dysfunction and injury severity scores, as well as lower lactate concentrations and base deficits, additional values that can be useful in monitoring the cardiovascular status of critically ill patients (see later in chapter for details).

A recent veterinary study evaluated the use of tissue perfusion parameters as predictors of outcome in dogs with severe sepsis or septic shock. $ScvO_2$ and base deficit (see next section) were found to be the best discriminators between survivors and nonsurvivors.[33]

There are some limitations to the measurement of SvO_2 and $ScvO_2$. Both hemoglobin concentration and SaO_2 influence these variables. $ScvO_2$ is much easier to measure, but there can be a loss of correlation between $ScvO_2$ and SvO_2 in very-low-flow states. And finally, if there is an underlying defect in oxygen extraction, as often occurs in patients with severe sepsis, the SvO_2 and $ScvO_2$ values can be normal or even high despite significant oxygen debt.[30]

LACTATE AND BASE DEFICIT

Lactate is produced primarily in periods of insufficient oxygen delivery to the tissues, during anaerobic glycolysis. It is produced from pyruvate by lactate dehydrogenase in the cytosol of cells. When oxygen balance at the cellular level is restored, the process is reversed; and the lactate is used for regeneration of pyruvate, and aerobic metabolism within the mitochondria is resumed. Normally, the liver (and to lesser degree, the kidneys) clears any lactate that is produced, but blood levels increase when production exceeds clearance (see Chapter 56).

Lactate levels at presentation as well as lactate clearance after treatment have been evaluated for prognostic value extensively in humans and to a lesser degree in veterinary patients.[30] Response to therapy, particularly fluid resuscitation, has been shown to have predictive value in humans with trauma and severe sepsis or septic shock. The base deficit has also been evaluated as a marker of anaerobic metabolism. In human studies, patients with persistently high base deficit have higher rates of multiple organ failure and death compared with patients whose base deficit normalizes. The use of lactate and base deficit together may be most helpful in assessing a patient's need for fluid resuscitation and response to fluid therapy. A recent study found that a lower base deficit at presentation was associated with greater survival in dogs with sepsis or septic shock secondary to pyometra.[33] As noted earlier, $ScvO_2$ and base deficit were found to be the best discriminators between survivors and nonsurvivors; lactate level was measured but did not prove useful in this study.[33] Additional studies are needed to prospectively evaluate the outcome prediction utility of lactate and base deficit response to volume resuscitation in dogs and cats.

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