Central venous pressure (CVP) is the hydrostatic or luminal pressure in the intrathoracic vena cava. Because CVP is affected by circulatory mean systemic pressure and venous return and because it affects cardiac preload, its measurement provides valuable information about cardiac performance and intravascular volume. CVP varies throughout both the respiratory and cardiac cycles; during inspiration, intrathoracic pressure decreases and CVP falls. In the presence of normally functioning cardiac valves, right ventricular cardiac preload (i.e., the amount of blood in the ventricle at the end of diastole) is affected by right atrial pressure, which in turn is determined by CVP. CVP therefore indirectly reflects right ventricular preload. Monitoring cardiac preload via CVP in critically ill patients can help ascertain blood volume status and subsequently direct the course of fluid therapy. It also helps clinicians interpret the cardiac response to intravenous fluids and thus identify which patients are likely to develop pulmonary edema, even before traditional clinical signs of fluid overload occur. In addition, measuring CVP can be helpful in determining the presence of volume depletion secondary to trauma or illness and can be used in the management of renal dysfunction, heart disease, septic shock, or any patient that may require administration of large volumes of intravenous fluids.

Measuring CVP is technically simple. The goal of monitoring CVP is to provide an objective assessment of both volume status and cardiac function in response to intravenous fluids. Monitoring CVP can help prevent volume overload from occurring and aid in volume resuscitation for critical hypovolemic patients. It can provide early evidence of volume overload in patients with suspected cardiovascular disease, pericardial effusion, or right-sided heart failure. Causes of elevated CVP not associated with volume overload include pleural or pericardial effusion, pulmonary hypertension, pulmonary thromboembolism, and pneumothorax; CVP may also be elevated in patients with poor pulmonary compliance or that are mechanically ventilated and require increased positive end expiratory pressure or increased mean airway pressures.

**DIAGNOSTIC CRITERIA**

**Historical Information**

**Age/Gender/Breed Predispositions**

- None. CVP monitoring can be used on any animal.
- CVP monitoring is particularly useful for patients at risk of volume overload (e.g., geriatric patients, oliguric patients with acute renal failure).
- CVP monitoring can be used to avoid volume overload.

**Also in this issue:**

6 Management of Canine Paraphimosis
CVP monitoring can be helpful in patients with septic shock because vasodilation contributes to hypotension despite seemingly adequate fluid loading.

CVP monitoring can help prevent fluid overload in patients with acute oliguric renal failure (urine output less than 1 ml/kg/hr).

Other Historical Considerations/Predispositions
Monitoring CVP is often indicated to help assess cardiovascular function. CVP is a measure of pressure generated in the intrathoracic vena cava as deoxygenated blood returns to the heart. If heart rate and myocardial function are held constant as venous return decreases, CVP decreases. Similarly, as venous return increases, CVP increases. This is of clinical importance as the CVP is used to measure the filling pressures of the right side of the heart, or more specifically, the right atrial and right ventricular end-diastolic pressures. Extreme elevations in CVP are seen with right-sided heart failure and pericardial effusion. Pleural effusion can also cause abnormally high CVP in the absence of suspected right-sided heart failure.

Monitoring CVP is useful in patients receiving large volumes of fluids, especially when physical examination is insufficient to assess an end-point for fluid resuscitation. Because CVP can be affected by cardiac function, CVP should also be monitored in patients with suspected heart disease.

CVP may be high as a result of conditions associated with decreased ventricular compliance, such as hypertrophic cardiomyopathy, pericardial effusion, and right AV valvular insufficiency.

CVP, like other hemodynamic markers, can be a useful tool only when used in combination with other parameters to assess critical patients. Note that CVP trends are more important than a single number. Typically, normal CVP values range from 0 to 8 cm H₂O, with values less than zero indicating hypovolemia and values over 10 cm H₂O suggesting volume overload.

In general, a low CVP value (less than 0 cm H₂O) is consistent with hypovolemia and may indicate insufficient blood volume in the ventricle during diastole or increased venous compliance secondary to septic shock or endotoxemia. The combination of low CVP values with tachycardia and other physical examination findings indicating poor perfusion (e.g., poor pulse quality or poor jugular filling, pale mucous membranes, prolonged capillary refill time) usually indicates the need for volume replacement.

Conversely, tachycardia or jugular distention combined with elevated CVP values may suggest fluid overload. A high CVP (greater than 8 cm H₂O) indicates volume overload, tricuspid valve insufficiency, increased pulmonary vascular resistance (afterload), pericardial effusion, or other causes of right-sided congestive heart failure. Patients with consistently elevated CVP readings exceeding 10 cm H₂O may develop edema and/or effusions.

Physical Examination Findings Associated with Abnormal CVP
- Low CVP measurements consistent with hypovolemia may accompany physical examination findings of poor perfusion, which include:
— Generalized weakness; dull mentation.
— Poor peripheral pulse quality; prolonged capillary refill time.
— Pale or muddy, dry mucous membranes.
— Abnormal respiratory rate and/or effort.
— Decreased urine output.

• Increased trends or elevated CVP measurements may indicate volume overload; clinical signs include:
  — Increased heart rate.
  — Increased respiratory rate.
  — Harsh lung sounds.
  — Chemosis.
  — Clear nasal discharge.
  — Increased body weight.
  — Jugular vein distention.
  — Peripheral edema.
  — Radiographic evidence of pulmonary edema, pleural effusion, and pulmonary venous congestion.

TREATMENT

Treatment of critical patients should not be based solely on CVP measurements; physical examination findings should always be weighed more significantly than CVP readings. In addition, edema and effusion can be secondary to other processes, such as increased capillary endothelial permeability associated with systemic inflammation and low oncotic pressure, even in hypovolemic patients.

• If the CVP is less than 2 cm H\textsubscript{2}O, real or functional hypovolemia should be suspected. A fluid challenge can be administered intravenously by a 10 to 15 ml/kg bolus of a crystalloid fluid or a 3 to 5 ml/kg bolus of a colloid solution. The CVP should be rechecked immediately after administration of the fluid bolus:
  — Animals with hypovolemia will show little or no change in CVP.
  — Animals with normovolemia will show a transient increase in CVP of 2 to 4 cm H\textsubscript{2}O with a return to baseline within 15 minutes.
  — Animals with hypervolemia or reduced cardiac compliance will show a sustained increase in CVP (greater than 4 cm H\textsubscript{2}O) that continues for more than 30 minutes.

• If the CVP exceeds 10 cm H\textsubscript{2}O, fluid therapy should be discontinued until CVP decreases; fluid rate should then be decreased. In some cases of volume overload, intravenous furosemide is indicated. Causes of elevated CVP include:
  — Volume overload.
  — Right-sided heart failure, tricuspid insufficiency, dirofilariasis, pericardial effusion or tamponade, and restrictive pericarditis.
  — Increased pulmonary vascular resistance (e.g., pulmonary hypertension, pulmonary fibrosis, heartworm disease).

CHECKPOINT

— Contraindications to measuring CVP are few and relate to placement of a central venous catheter. Patients that may have complications with central venous catheterization include those with a history of:
  • Coagulopathies, which may cause excessive bleeding from the venipuncture site.
  • Thromboembolic diseases.
  • Hyperadrenocorticism.
  • Immune-mediated hemolytic anemia.
  • Increased intracranial pressure (e.g., because of head trauma, seizures, intracranial disease).
  • Respiratory distress.

Procedural Recommendations

Equipment $\
• Central venous catheter (Arrow International; 18-gauge for cats, 16-gauge for medium-sized dogs, 14-gauge for large-breed dogs; commercial length typically 20 cm).
• T-port (Arrow International).
• Manometer (Allegiance or Cardinal Health).
• Extension tubing.
• Three-way stopcock.
• Saline-filled syringe.

Technique

The use of standard protocols in measuring CVP is important to minimize infection, to promote catheter longevity, and most importantly, to yield consistent results. Recommended technique:

• Tip of a central venous catheter should be located in the cranial vena cava just outside the right atrium.
• Patient should be in right lateral recumbency with the head and neck hyperextended to avoid positional catheter issues.
• The saline-filled syringe, saline-flushed extension set, and three-way stopcock are attached to the manometer.
• The central venous catheter is attached to the manometer via the extension set.
• The zero point on the manometer should be equal with the level of the patient’s heart; the zero point can be gauged visually by lining it up with the manubrium (most cranial portion of the sternum).

• The stopcock is turned off to the patient and the manometer filled with saline. The stopcock is then turned off to the syringe, allowing direct communication between the manometer and the catheter. The saline column is allowed to equilibrate. Proper placement of the catheter will result in small fluctuations of the fluid meniscus coinciding with changes in intrathoracic pressure that occur with respirations.

• CVP equals the level of the fluid on the manometer less the number correlating with the zero point. For example, if the fluid meniscus is at 15 cm H$_2$O and the zero point (manubrium) is at 10 cm H$_2$O, the CVP is estimated to be 5 cm H$_2$O.

• Several readings should be taken before recording a measurement.

Practical Tips
• Because CVP values can be affected by a patient’s position, consistency in patient positioning is critical.

• Incorrect catheter placement affects readings; placement should be confirmed by radiography or fluoroscopy.

• Changes in normal intrathoracic pressures (e.g., because of pneumothorax or positive pressure ventilation) will affect CVP readings, resulting in falsely high readings.

• Undulations in fluid level that correlate to the patient’s heartbeat may indicate excessive length of catheter in the right atrium or right ventricle and will result in falsely elevated CVP measurements.

• Placement of a multilumen catheter allows for concurrent fluid therapy, drug infusions, and/or blood sampling as well as CVP monitoring. CVP values should be measured through the distal port (usually the colored port) if a multilumen catheter is used.

• In slowly evolving fluid overload with fluid therapy (hours to days), CVP may not increase until the capacitance veins have reached maximum distension and edema is imminent; therefore, CVP monitoring is less valuable than body weight change and other physical findings in this setting. CVP will change much more rapidly and unequivocally in response to rapid fluid challenges.

• A catheter inserted into the femoral vein can also be used to estimate CVP. The tip of the catheter should be positioned in the abdominal vena cava, which will provide a good estimate of right ventricular filling pressure as long as there is not increased abdominal pressure from bladder distention, an abdominal mass, severe abdominal pain, or an abdominal pressure wrap.

• Sterility should be emphasized both during catheter placement and when taking each CVP reading. Nursing care becomes particularly important because central catheters are typically left in place for a longer time than are peripheral catheters. Daily inspection of insertion site, daily bandage changes, intermittent flushing with heparinized saline, and conscientious practice of sterile technique during measurement readings are of utmost importance. Excessive loops of tubing should be avoided, Luer-lock connections should be used to prevent fluid leakage or air boluses, and three-way stopcocks on the catheter itself are needed to avoid introduction of bacteria; likewise, manometers need to be taped onto the cage or stored with sterile caps if used intermittently.

Prognostic Criteria
Favorable
• CVP measurements in the normal range combined with normal urine output (more than 2 ml/kg/hr) in patients with acute renal failure.

• CVP values returning to the normal range after discontinuation of intravenous fluids or administration of furosemide in patients showing signs of hypervolemia.

• Negative or low CVP readings increasing to 5 to 7 cm H$_2$O in response to fluid loading, pressor therapy, or positive inotrope therapy in patients with vasodilatory shock.

• Decrease in CVP readings to normal range following pericardiocentesis in animals with pericardial effusion.
Unfavorable

- Increasing CVP in patients with acute renal failure and anuria or oliguria is a poor prognostic indicator.
- Volume overload associated with cyanosis, respiratory distress, and pulmonary edema has a guarded prognosis and should be corrected immediately and the patient treated with supplemental oxygen.
- Acute increase in CVP associated with labored breathing can be seen with pulmonary hypertension secondary to pulmonary thromboembolism. Prognosis is guarded.
- Gradually increasing CVP and progressive dyspnea can also be seen with accumulating pleural effusion. Thoracentesis is indicated.

RECOMMENDED READING

