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Compendium

Central Venous Pressure: Principles, Measurement, and Interpretation

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Abstract: *Central venous pressure (CVP), an estimate of right atrial pressure, has been used to assess cardiac preload and volume status in critically ill patients, assist in the diagnosis of right-sided heart failure, and guide fluid resuscitation. It is determined by the interaction between cardiac function and venous return. CVP measurements are relatively easy to obtain; however, because of the complex relationship between CVP, cardiac output, and the vascular system, they may be difficult to interpret. This article reviews the physiologic principles of CVP, indications for its use, technical aspects and pitfalls of CVP measurements, and interpretation of the information obtained.*

One of the most important goals in the management of patients with perfusion abnormalities is to optimize preload through volume resuscitation.[1] While direct measurements of ventricular end-diastolic volume allow for the most accurate assessment of preload, they are not always practical in a clinical setting.[2] Consequently, pressure measurements such as central venous pressure (CVP) and pulmonary artery occlusion pressure have been traditionally used as substitutes for volume estimates.[2]

CVP has been considered a reliable indicator of right ventricular preload.[2] It has been used to assess cardiac preload and volume status in critically ill patients,[3] assist in the diagnosis of right-sided heart failure, and help guide fluid resuscitation.[4] It has also been advocated as a component in the assessment of adequate fluid resuscitation in patients with septic shock.[1] However, the utility of CVP as a measure of right ventricular preload in people has been questioned.[2,5–8] In human and veterinary clinical practice, CVP is frequently used to gain information about cardiac function and intravascular volume.[1,9] Despite the common use of this measurement, its physiologic meaning and clinical application are frequently misunderstood.

Physiologic Principles

CVP is the blood pressure within the intrathoracic cranial or caudal vena cava relative to atmospheric pressure.[8] Physiologically, however, ventricular preload is determined by transmural pressure, which is the difference between intracardiac and extracardiac intrathoracic pressure.[10] Therefore, while an increase in intrathoracic or intrapericardial pressure may cause a decrease in venous return and subsequently a decrease in preload, it may also lead to an increase in the CVP. Multiple factors influence transmural pressure, including positive-pressure ventilation, cardiac tamponade, pleural effusion, and increased intraabdominal pressure.[3,10,11]
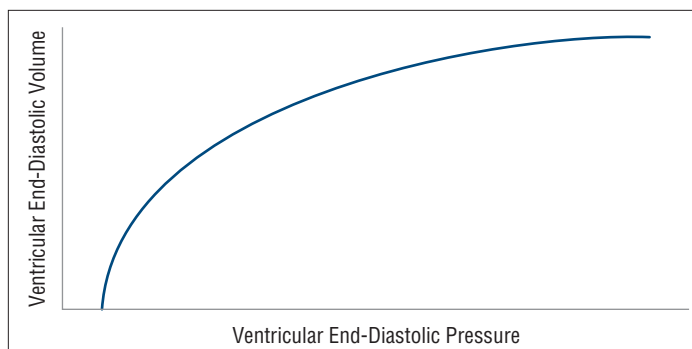

Figure 1. This graph depicts the relationship between ventricular end-diastolic pressure and ventricular end-diastolic volume (Frank–Starling relationship of the heart). As pressure increases, the ventricular filling volume increases rapidly until the limit of ventricular distention is reached. Beyond this point, even large increases in pressure will not significantly increase ventricular volume.[2]

The use of the cranial versus the caudal abdominal vena cava for measurement of CVP has been investigated in puppies,[12] infants, and children,[13] and studies show that there may be a correlation between measurements taken from centrally placed catheters in the cranial intrathoracic and caudal abdominal vena cavae in cats,[14] puppies,[12] and children.[13] However, it has also been demonstrated that intraabdominal hypertension causes an increase in right atrial pressure and, therefore, CVP.[15] Intraabdominal hypertension also causes increases in pulmonary artery pressure, systemic vascular resistance, and pulmonary capillary wedge pressure. These changes, along with increased CVP, are attributed to catecholamine release and vascular shifts from the abdominal to the thoracic cavity.[15]

Common conditions that may lead to abdominal hypertension include pancreatitis, bile peritonitis, intraabdominal hemorrhage, large abdominal masses, blunt abdominal trauma, recent abdominal
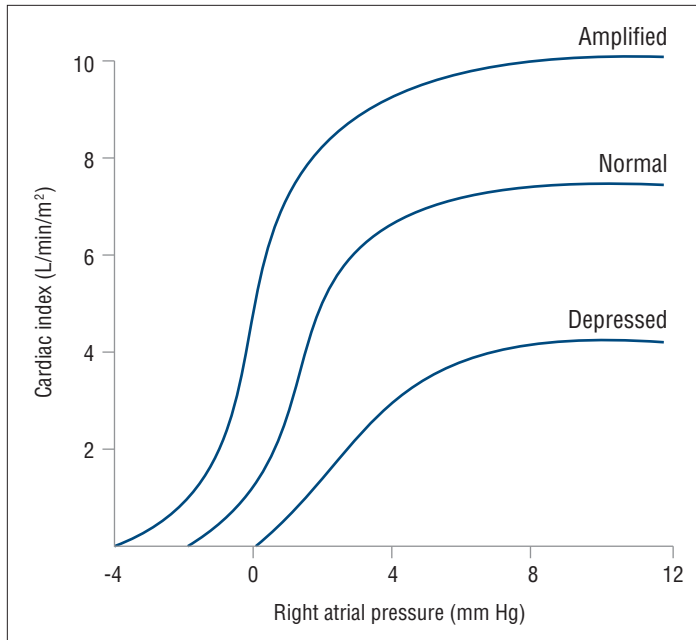
Figure 2. The relationship between right atrial pressure (or its surrogate, CVP) and cardiac output (L/min), represented here by the cardiac index (L/min/m²), is not linear. At low pressures, small increases in CVP may indirectly lead to large increases in cardiac output via increases in stroke volume. As the curve plateaus, at high right atrial pressures, even large increases in right atrial pressure (CVP) have little effect on cardiac output.[2]

surgery, and fluid resuscitation.[16] Despite the correlation demonstrated between measurements taken in the cranial intrathoracic and caudal intraabdominal vena cavae,[12] measurements taken from the intrathoracic vena cava are preferred. The general recommendation for small animal patients[9,17] and human pediatric patients[18] stipulates that the catheter tip lie within the thoracic cavity when CVP measurements are made.

CVP provides an estimate of right atrial pressure[4,8,9,19] and is used as a surrogate for right ventricular volume at the end of diastole. Right ventricular end-diastolic volume is proportional to preload, which is the major determinant of stroke volume at any given level of contractility.[8] Cardiac output is determined by heart rate and stroke volume, and stroke volume is determined by preload, afterload, and contractility. The relationship between ventricular end-diastolic volume and pressure is not linear, but curvilinear (**FIGURE 1** and **FIGURE 2**). Increasing ventricular end-diastolic volume (or its surrogate, CVP) by intravenous fluid administration will, according to the Frank-Starling law, initially lead to increases in stroke volume and therefore cardiac output. Eventually, however, further fluid administration leads to increases in end-diastolic pressure, but no substantial increases in end-diastolic volume or stroke volume.[4,20]

CVP is determined by the interaction between cardiac function and venous return,[3] which in turn are influenced by total blood volume, vascular tone, cardiac output, right ventricular compliance, and intrathoracic pressure. Right ventricular compliance may be altered by myocardial and pericardial disorders.[8] Ventricular

compliance (the change in volume for a given change in pressure) shows significant individual variability in health and in disease states in people.[2] Ventricular end-diastolic pressure provides an accurate reflection of end-diastolic volume only when ventricular compliance is normal. Therefore, CVP cannot be assessed, or preload inferred, if compliance of the ventricle is abnormal. Decreased right ventricular compliance can increase the CVP. Pathologic processes leading to decreased right ventricular compliance include disorders that increase myocardial stiffness, including ischemia, infiltrative diseases, and fibrosis. Other causes of decreased right ventricular compliance include conditions that lead to myocardial hypertrophy, such as pulmonic stenosis, pulmonary hypertension, and pericardial disorders such as pericarditis and pericardial effusion.[9,17]

CVP has also been used as a surrogate for left ventricular end-diastolic volume.[10] As the right side of the heart and lungs act as a passive conduit for blood, left atrial pressure and CVP may be related in healthy animals. However, in critically ill patients, or those with heart disease, assumptions about the left heart based on CVP measurements may be incorrect.[21] Measurements of right ventricular filling pressures are associated with interpretive problems in critically ill people,[22] leading to the argument that such measurements do not accurately reflect filling pressures of the left ventricle.[23] Any anatomic, physiologic, or pathophysiologic factor affecting the pressure between the left ventricle and the right atrium can alter the relationship between the measured CVP and the left ventricular end-diastolic volume. Examples include tricuspid valve disease, changes in right or left ventricular compliance, pulmonary hypertension, pulmonary venous disease, chronic airway disease, and mitral valve disease.[22] Therefore, a pressure reading from the right atrium does not necessarily reflect volume in the left ventricle. However, CVP can be used to estimate left ventricular volume via right atrial pressure as long as it is understood that many factors may interfere with this measurement.

Principles of Measurement

The animal may be positioned in right lateral, left lateral, or sternal recumbency; lateral recumbency is preferred. A study by Oakley and colleagues[24] in healthy dogs found no differences in CVP measurements obtained from animals in left or right lateral recumbency. For measurement consistency, the patient should be placed in the same position for each CVP measurement.[17]

The CVP can be measured using an electronic pressure transducer or a water column manometer. In contrast to electronic pressure transducers, water manometers may overestimate the mean CVP by 0.5 to 5 cm H_2O.[4] These overestimations are partly due to a meniscus effect of the water column in the manometer and partly due to observer estimation of a "mean" CVP value that naturally fluctuates during inspiration and expiration.[25] Overestimations often vary from patient to patient or from reading to reading in the same patient even with careful positioning.[9] In critically ill patients, continuous evaluation of CVP using an electronic pressure transducer connected to the central venous catheter by a short stiff tube may be more accurate in assessing rapidly changing trends and responses to fluid boluses.[9,25]
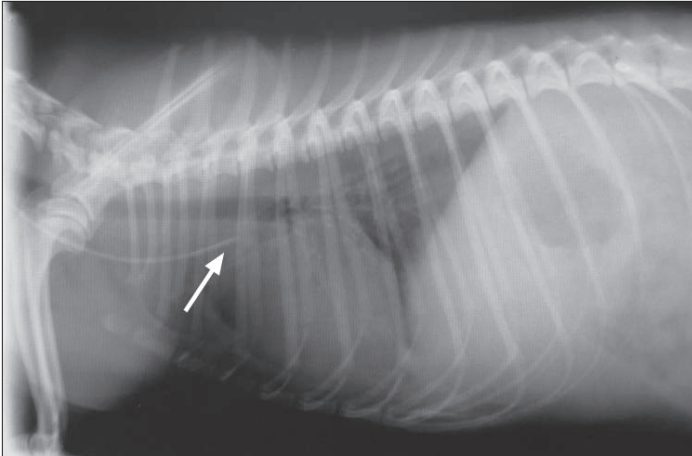
Figure 3. Radiograph of appropriately positioned central line (*arrow*).
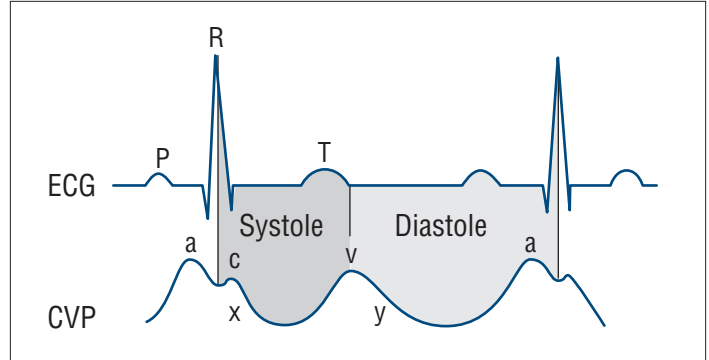

Figure 4. Normal CVP waveform with normal electrocardiography tracing. Central venous pressure tracings show three positive waves (a, c, and v) and two descents (x and y). These waveforms are a reflection of slight variations in CVP during the different phases of the cardiac cycle. The a wave represents an increase in right atrial pressure during atrial contraction. The c wave represents an increase in right atrial pressure due to bulging of the tricuspid valve into the right atrium during early ventricular systole. The x descent represents a reduction in right atrial pressure during ventricular ejection. The v wave represents an increase in right atrial pressure during rapid atrial filling when the tricuspid valve is closed. The y descent represents a decrease in right atrial pressure after the tricuspid valve opens and there is rapid filling of the right ventricle. *Adapted from Central venous pressure waveforms. University of Virginia Health System. Accessed February 2010 at http://www. healthsystem. virginia.edu/internet/anesthesiology-elective/cardiac/cvpwave.cfm.*[27]

In fluid-filled systems, hydrostatic pressure measurements are made relative to an arbitrary reference point, and changes in that reference point affect the measured pressure.[26] A commonly accepted reference point for measurement of the CVP is the midpoint of the right atrium, where the blood returning to the heart interacts with cardiac function.[26] To obtain a CVP measurement, a central catheter should be placed in the cranial intrathoracic vena cava with the catheter tip located just outside the right atrium.[9] Proper intraluminal placement of the central catheter, without obstruction against the vessel wall, can be confirmed by the ability to draw blood without difficulty.[9] Contact with the endocardium of the right atrium or ventricle may stimulate cardiac arrhythmias.[20] Thoracic radiographs should be obtained to confirm appropriate catheter position (**FIGURE 3**).

The presence of larger fluctuations in the height of the meniscus fluid column, which mimic the animal's respiration, and smaller fluctuations mimicking the heartbeat may also help in assessing proper catheter placement.[9,17] The rhythmic fluctuations seen with respiration occur because the intrathoracic pressure and, therefore, CVP change during inhalation and exhalation.[9] Catheters inappropriately positioned within the right ventricular lumen produce high CVP readings with large fluctuations of the fluid meniscus synchronous with the heartbeat.[9]

Electronic monitors and pressure transducers can be used to measure CVP continuously. Electronic pressure tracings contain identifiable waveforms that cannot be detected with a water manometer.[9] These waveforms are a reflection of slight variations in CVP during the different phases of the cardiac cycle and, when used in conjunction with electrocardiography, can be used to obtain measurements at the most appropriate time of the cardiac cycle[27] (**FIGURE 4**). The pressure at the base of the c wave represents the final right atrial pressure at the start of systole and is, therefore, the best estimate of right ventricular preload.[26] CVP measurements should be taken at the end of exhalation, when intrathoracic pressure is only slightly negative relative to atmospheric pressure.[26] The ability to use waveforms for interpretation of CVP is limited to electronic pressure transducers because the

response of the fluid column in water manometers is too slow to demonstrate the pressure changes accurately.[9]

FIGURE 4 shows the normal CVP waveform in a person. The c wave is produced at the onset of ventricular systole as the tricuspid valve closes and bulges into the right atrium. This wave is small and inconsistent and is often not seen in veterinary medicine.[19,28] When the c wave is not readily evident, the base of the a wave may be used to obtain a similar estimate of right ventricular preload. The ECG tracing taken concurrently with the CVP measurement can also be used to time and assess the beginning of systole.[29] As ventricular ejection continues, the annulus moves toward the cardiac apex, which leads to a drop in right atrial pressure, producing the x descent.[28] Ventricular ejection drives venous return through the vena cava, which increases right atrial volume and pressure during mid to late systole (at or after the T wave). Right atrial pressure increases as the right atrium fills against a closed tricuspid valve, producing the v wave.[10,28] The subsequent y descent represents the decrease in atrial pressure as the tricuspid valve opens and the right atrium empties blood into the right ventricle (in the T–P interval).[28] The last part of diastole is marked by atrial contraction, which increases the right atrial pressure to its highest value, represented by the a wave (in the P–R interval).[19,28]

Measurement of Central Venous Pressure Using a Water Manometer

To measure CVP with a water manometer, the following materials are recommended[9] (**FIGURE 5**):

- Central venous catheter
- Water manometer
- 30-inch IV extension set

Figure 5. Supplies needed for water manometer (Medex manometer set, Smiths Medical, London). Not shown: central venous catheter, 20-gauge needle, and string or tape measure.

- Three-way stopcock
- 6- to 20-mL syringe filled with saline
- String or tape measure

Alternatively, a sterile bag of fluids may be used instead of the syringe.[17]

After the patient is placed in the desired position and the catheter is appropriately positioned, connect the water manometer in series with the central venous catheter and the syringe or fluid bag. With the stopcock turned "off" to the manometer, prime the fluid line and the extension set with fluid to remove any air bubbles. In order to make the measurement more reproducible, the stopcock should rest on the floor or the bottom of the cage (**FIGURE 6**). Assess the patency of the catheter by allowing fluid from the syringe or saline bag to flow freely into the catheter. If a multilumen central catheter is used, infusions of fluids through other lumens should be discontinued before obtaining a CVP reading, as they may artificially increase the measurement.[30]

Before CVP can be measured with a water manometer, identify the zero point on the manometer that corresponds to the patient's right atrium. This measurement will need to be subtracted from the reading to obtain the correct CVP. When the patient is in lateral recumbency, the sternum serves as a reference point for the right atrium. For a patient in sternal recumbency, the scapulohumeral joint or the point of the shoulder is a good reference point. To find the zero point on the manometer, draw a perpendicular line from the reference point on the patient to the manometer using the string or tape measure (**FIGURE 6**). For a small dog, the zero point is usually 2 to 3 cm H_2O. Therefore, if the column of water in the manometer equilibrates at 10 cm H_2O and the zero point is 2 cm H_2O, the actual CVP would be 8 cm H_2O.

To obtain a measurement, fill the manometer with saline significantly above the expected patient CVP. Turn the three-way stopcock so that the column of saline in the manometer is continuous with the central catheter and the stopcock is "off" to the saline bag or syringe. The saline in the manometer will decrease until the hydrostatic pressure in the column equilibrates with the hydrostatic pressure of the blood at the tip of the central catheter. Once the saline in the manometer stops falling, it has reached the
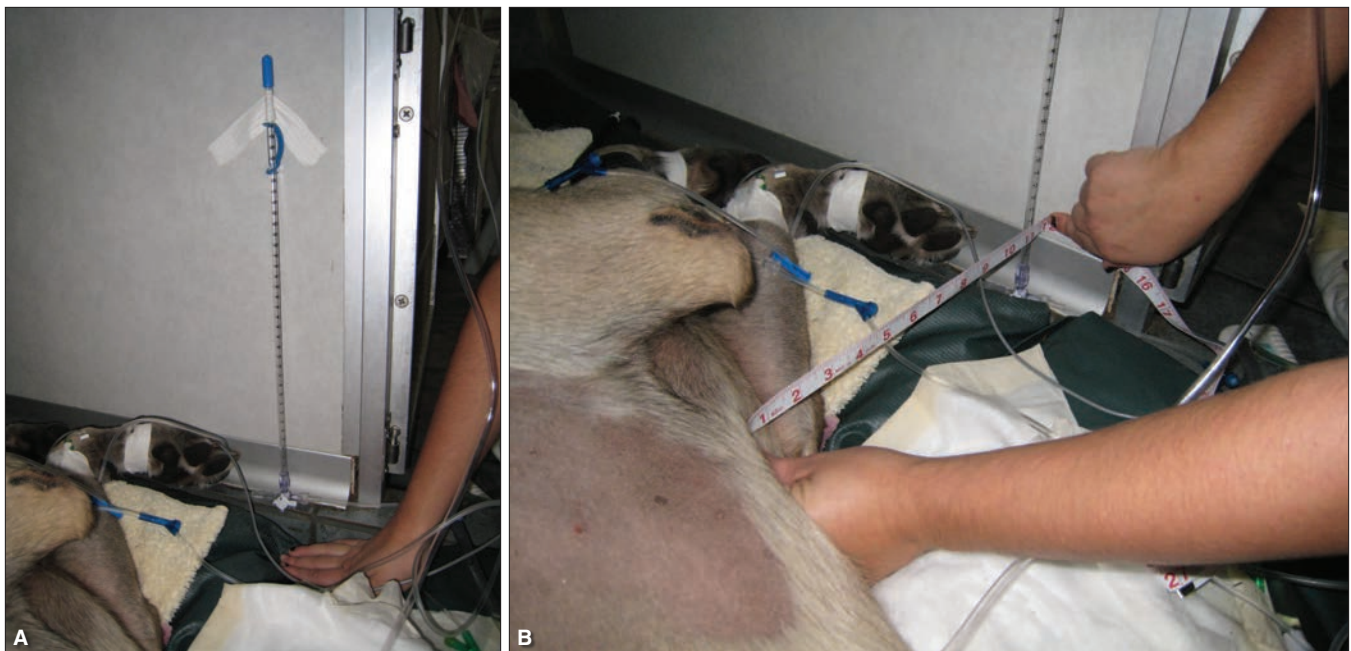

Figure 6. (**A**) Measurement of CVP using water manometer. (**B**) Perpendicular line from the patient to the manometer denoting zero point.
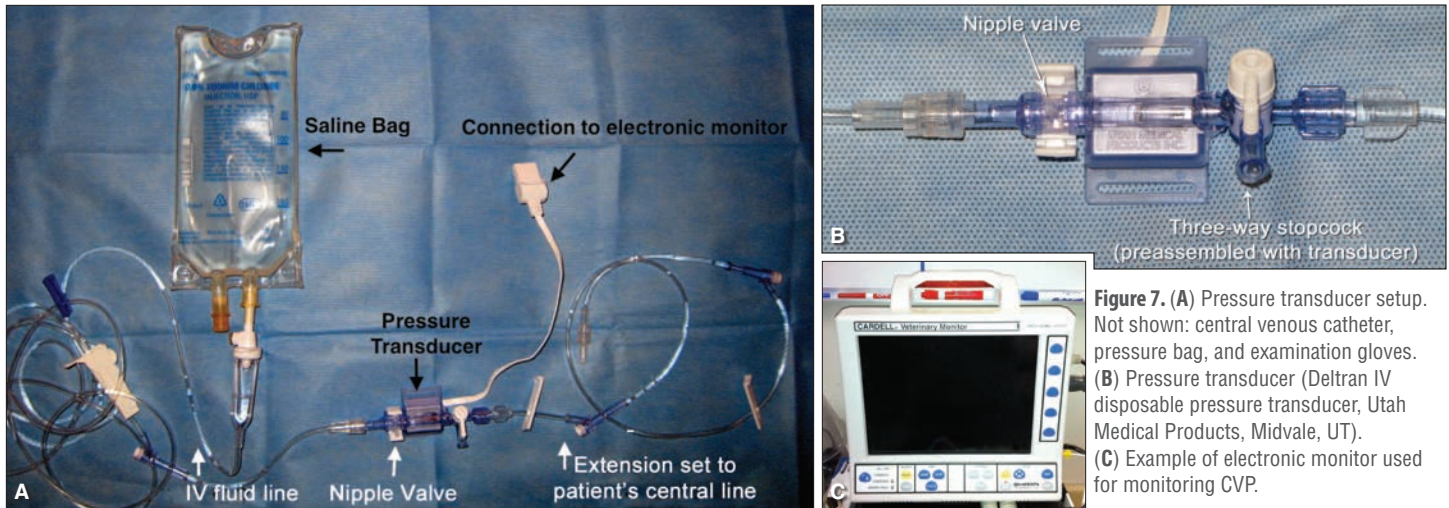
Figure 7. (**A**) Pressure transducer setup. Not shown: central venous catheter, pressure bag, and examination gloves. (**B**) Pressure transducer (Deltran IV disposable pressure transducer, Utah Medical Products, Midvale, UT). (**C**) Example of electronic monitor used for monitoring CVP.

equilibrium point, which reflects the blood pressure inside the vessel at the point of the catheter tip.[9] The measurement is expressed in cm H_2O. Normal reference ranges in dogs and cats are reported to be 0 to 10 cm H_2O.[17,30]

Measurement of CVP with an Electronic Pressure Transducer

To obtain a measurement with an electronic pressure transducer, the following materials are recommended[4,17] (**FIGURE 7**):

- Central venous catheter
- Electronic monitor capable of recording pressures
- Pressure transducer with flush valve
- 8- or 12-inch arterial blood pressure tubing setup or extension set
- Pressure bag
- Bag of saline
- Examination gloves
- Drape

Connect the pressure transducer to the patient monitor before turning it on. Wearing the examination gloves, cover a clean horizontal surface with the drape and assemble the setup. Typically, transducers have a flush nipple valve, which is attached to a pressurized bag of saline to facilitate flushing of the system.[4,31] Position the transducer in line with the patient's central catheter using flexible but fairly rigid-walled tubing. Arterial blood pressure tubing works well for this purpose. The transducer converts the mechanical energy of the pressure waves into electric signals[19] that are amplified and displayed or recorded. It is important to use rigid-walled, short tubing because less stiff tubing has greater compliance and may dampen the waveform.[10] This is not a problem with water manometers because the height of the ball or meniscus in the manometer is a direct reflection of the pressure in the right atrium, and no conversion of mechanical energy into an electrical signal is necessary.[10]

Before attaching the system to the patient, purge the system of air and prime the line by opening the stopcock to room air and flushing saline through the line. Even small air bubbles can dampen

the CVP waveform and cause an underestimation of CVP.[31] Also, zero the transducer and monitor by turning the stopcock so that the system is open to room air and by pressing the "zero" button on the monitor until it reads (0). The scale on the monitor should be adjusted to the lowest range, typically 18 to 22 mm Hg (25 to 30 cm H_2O).

Turn the stopcock and pull the nipple valve to allow the saline to run through the system into the central catheter to verify catheter patency. The transducer must be positioned at the level of the catheter tip. The landmarks for placement of the transducer are the same as those used for measuring CVP with a water manometer. Finally, turn the stopcock so that the transducer is continuous with the patient's catheter to obtain a reading.

Measurements are expressed in systolic, diastolic, and mean CVP, and the mean CVP is used.[4] CVP measurements obtained with electronic pressure transducers are usually displayed in mm Hg. To convert a measurement in mm Hg to cm H_2O, multiply the value by 1.36. If respiration causes significant fluctuation of the baseline, it is recommended to measure the CVP from a printout or calibrated screen, using the base of the c wave at end-expiration.[4]

Interpretation

CVP has been used to assess cardiac preload and volume status,[3,26] to optimize right ventricular preload,[2] and to monitor fluid therapy in an attempt to minimize the risk of volume overload in patients with oliguric renal failure or heart failure.[9] In addition, CVP has been advocated as a means to monitor fluid resuscitation in patients with noncardiogenic shock[1,4] and to assess patients for recurrent pericardial effusion and right-sided heart failure.[4,19] A survey of human criticalists showed that many relied on cardiac filling pressures such as CVP and pulmonary artery occlusion pressure to help guide fluid therapy.[32]

Accurate assessment of preload and volume status is vital to the appropriate management of critically ill patients.[9] Unfortunately, direct measurement of right and left ventricular end-diastolic pressures requires the use of invasive techniques such as placement

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Box 1. Factors Affecting Measured Central Venous Pressure[16,29,35]

Technical factors
- Patient positioning
- Level of transducer
- Inconsistent measurement technique with water manometer
- Inappropriate central venous catheter placement
  —Against vessel wall
  —In heart

Physiologic factors
- Changes in intrathoracic pressure
  —Respiration
  —Positive end-expiratory pressure during mechanical ventilation
  —Abdominal hypertension
- Changes in central venous blood volume
  —Total blood volume
  —Venous return/cardiac output
- Cardiac rhythm disturbances
  —Junctional rhythm
  —Atrial fibrillation
  —Atrioventricular dissociation

Changes in right ventricular compliance
- Myocardial stiffness
- Pericardial disease
- Cardiac tamponade

Tricuspid valve disease
- Tricuspid regurgitation
- Tricuspid stenosis

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of a thermister-tipped Swan-Ganz catheter.[9,33] These catheters are rarely available in veterinary medicine. Other methods, such as echocardiography, may be used to assess preload[6] by measuring left ventricular end-diastolic indices, but to our knowledge, these methods have not been validated in veterinary medicine. Right ventricular end-diastolic pressure estimated by CVP has been used as a substitute for assessing preload. Inherent in the use of CVP is the assumption that it is an acceptable substitute for right ventricular end-diastolic volume as an indicator of preload. This assumption may be flawed given the numerous conditions that can affect the measurement[34] (**BOX 1**).[16,29,35]

Studies in different human patient groups as well as healthy human volunteers found no predictable relationship between CVP and cardiac performance or between indices of right ventricular volume and stroke volume.[2,5–8] In addition, there was no significant correlation between an increase in CVP and changes in right ventricular volume and cardiac performance or stroke volume secondary to fluid therapy.[2,5,8] The lack of correlation between pressure and volume indices of preload has been largely

attributed to variations in ventricular compliance, which can be affected by trauma, infarction, sepsis, mechanical ventilation, positive end-expiratory pressure, and impaired diastolic relaxation due to ischemia, myocardial hypertrophy, or pharmacologic agents.[2] Even in healthy people, there seems to be a marked variability in right ventricular compliance.[2] The poor correlation between CVP as an estimate of right ventricular end-diastolic volume and cardiac performance should be considered when using CVP to estimate right ventricular preload.[4] Despite its shortcomings, CVP is one of the few available methods of assessing preload at the bedside. We do not feel that studies showing poor correlation between CVP and right end-diastolic volume preclude the use of CVP for assessment of preload, just that it must be used with these limitations in mind.

In general, low CVP values have been assumed to indicate hypovolemia[3,36,37] and high CVP values to indicate hypervolemia or congestive heart failure. However, neither reliably predicts the cardiovascular response to a fluid challenge.[1,36,38] Isolated CVP measurements are of limited value.[19] Instead, trends of CVP measurements over time or changes in response to a fluid challenge may provide more reliable information regarding intravascular volume status.[19,34] Rapid administration of a fluid bolus to a hypovolemic patient with good ventricular function should cause an increase in CVP of <2 mm Hg. A return to baseline CVP within 10 minutes, combined with improvement in blood pressure or other indicators of perfusion, suggests that the patient is volume responsive. However, if bolus administration results in a large increase in CVP (≥2 mm Hg) that persists without improvement in perfusion parameters, the patient is likely not volume responsive.[10]

Because not every patient may tolerate fluid challenge, Magder et al[39] attempted to define an accurate CVP value in human patients above which additional fluid challenges would not lead to increases in cardiac output, as determined by a cardiac index >300 mL/min/m^2. This study found that some patients failed to respond to fluid boluses at all levels of baseline CVP measurements, even those with CVP <3.7 mm Hg (5 cm H_2O). The study also found that a CVP >7.4 mm Hg (10 cm H_2O) was considered to be high and that the likelihood of a change in cardiac index secondary to a fluid bolus was low.[37] The investigators concluded that in people, a CVP of >7.4 mm Hg (10 cm H_2O) was a reasonable threshold for the upper limit of volume responsiveness. Studies of this kind have not been performed in veterinary medicine, and absolute high and low CVP values are very difficult to define. In addition, there may be variation between species. In general, low CVP values may indicate that additional intravenous volume loading may be beneficial and high values make continued volume expansion detrimental.[39,40]

Central Venous Pressure in Clinical Practice

CVP is determined by the interaction of cardiac function and venous return.[3,39] A high CVP may be detected in patients with normal cardiac function and increased intravascular volume (e.g., volume overload) or in patients with decreased cardiac function and normal intravascular volume (e.g., myocardial failure).
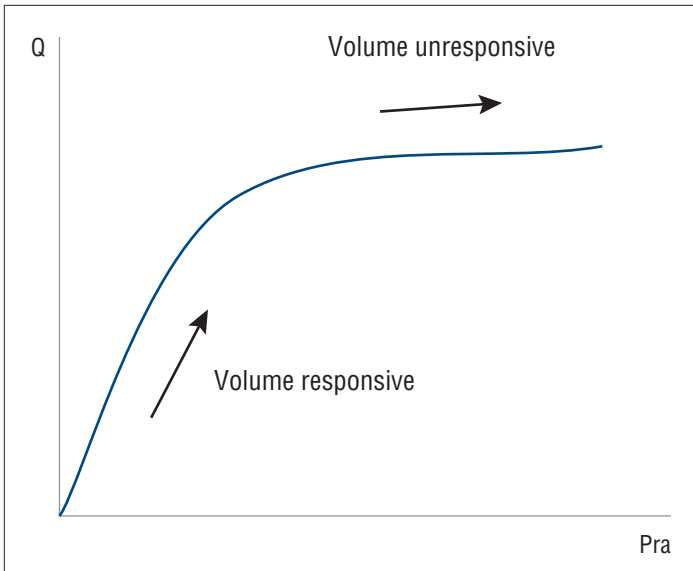
Figure 8. Fluid responsiveness demonstrating Starling's law. In the volume-responsive portion of the curve, a change in CVP will produce a measurable change in cardiac output. Once the plateau phase is reached, additional fluids will no longer increase cardiac output.[1] *Pra* = right atrial pressure, *Q* = cardiac output.
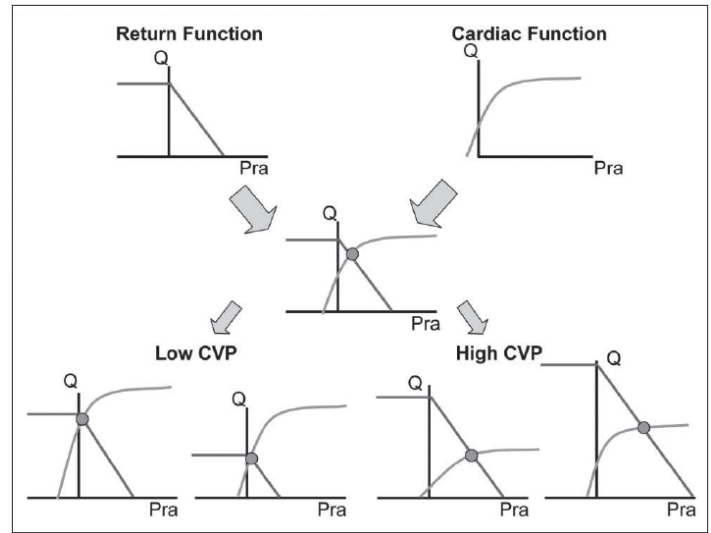

Figure 9. Interaction of the return function and cardiac function curves for the determination of right atrial pressure (Pra) or its surrogate, CVP, and cardiac output (Q).[3] The graphs in the lower left corner show low CVP with high cardiac function and normal blood volume and return function or with normal cardiac function but decreased volume. The graphs in the lower right corner show that a high CVP may be associated with normal return function but decreased cardiac function or normal cardiac function with high return function because of excess volume. This explains why a single CVP value alone may not explain cardiac function or volume status.[3] *Reproduced with permission from Magder S. How to use central venous pressure measurements.* Curr Opin Crit Care *2005;11:264-270.*

Conversely, CVP may be decreased in an individual with increased cardiac function and normal blood volume (e.g., during exercise) or in a patient with normal cardiac function and decreased blood volume (e.g., severe blood loss).[30] Thus, the clinical significance of a specific CVP measurement can only be assessed in conjunction with a measurement of cardiac output or a surrogate thereof[34] (**FIGURE 8**).[3] Cardiac output can be measured directly by thermodilution techniques using a Swan-Ganz or pulmonary arterial catheter, or indirectly by assessing parameters of perfusion such as mucous membrane color, capillary refill time, skin temperature, urine output, blood pressure, lactate, or central venous oxygen saturation.[1,9,36] In veterinary medicine, changes in perfusion parameters are used more commonly than Swan-Ganz catheters to assess cardiac output in conjunction with changes in CVP.

The gold standard for evaluating a patient's fluid responsiveness is to assess the hemodynamic response to a fluid challenge.[3] The decision of which type of fluid to be used—colloid or crystalloid—is based on the underlying illness. As a general guideline, a volume sufficient to raise the CVP by 1.5 mm Hg (2 cm H_2O) should be given as a bolus. It should be administered as quickly as possible because fluid given slowly will be accommodated by venous capacitance vessels and therefore may not lead to changes in CVP.[4] The change in cardiac output in response to the fluid bolus should be immediate.[3] The combination of an increase in CVP and an increase in cardiac output or improvement in perfusion indicates that the patient is fluid responsive. An increase of the CVP without an increase in cardiac output or an improvement in perfusion suggests that additional volume loading is not indicated because the patient is not fluid responsive[10] (**FIGURE 9**).[3]

For example, a patient with gastric dilatation-volvulus may show signs of hypoperfusion (e.g., gray mucous membranes, prolonged capillary refill time, tachycardia, decreased blood pressure) and have a low baseline CVP. After a bolus of crystalloids, the CVP may increase, and mucous membrane color, capillary refill time, heart rate, and blood pressure may improve. On the other hand, a fluid bolus given to a patient with congestive heart failure may lead to an increase in CVP but no improvement in perfusion parameters.

In veterinary medicine, a rapid infusion of 20 mL/kg of a crystalloid or 5 mL/kg of a colloid in a euvolemic animal with appropriate cardiac function may lead to a mild increase in the CVP (2 to 4 cm H_2O) that returns to baseline within 15 minutes. A minimal or absent increase in CVP suggests markedly reduced intravascular volume and the need for continued fluid resuscitation. A CVP that increases and returns to baseline within 5 minutes also suggests decreased intravascular volume and the need for continued fluid resuscitation. A large increase in CVP (>4 cm H_2O) suggests decreased cardiac function, increased intravascular volume, or a combination of the two. A prolonged return to normal (>30 minutes) may indicate increased intravascular volume.[9] Additional fluids could be detrimental in this case.

With these guidelines in mind, clinicians should recognize that the use of CVP to assess preload and fluid responsiveness is far from infallible. In studies designed to assess fluid responsiveness in critically ill human patients, only 40% to 72% of patients responded to volume expansion as demonstrated by a significant increase in stroke volume or cardiac output.[5] This lack of fluid responsiveness was attributed to high venous compliance, low ventricular compliance, and/or ventricular dysfunction.[5] Therefore,

Figure 10. (**A**) A normal CVP tracing. (**B**) A CVP tracing showing a cannon a wave (*asterisk*), which is produced when atrial contraction occurs against a closed tricuspid valve. *Reproduced with permission from Barbeito A, Mark JB. Arterial and central venous pressure monitoring.* Anesthesiol Clin *2006;(24)717-735.*

Figure 11. A CVP tracing showing giant v waves (ventricularization) caused by tricuspid regurgitation. The *arrows* represent the best place to estimate the mean CVP in this situation before the onset of the c-v regurgitant wave. Note the elimination of the x descent. Pressure generated during the right ventricular contraction is detected at the catheter tip.[10,28] *Reproduced with permission from Barbeito A, Mark JB. Arterial and central venous pressure monitoring.* Anesthesiol Clin *2006;(24)717-735.*

while CVP can be and has been used to assess preload and volume responsiveness, it may not always be reliable. Clinicians must recognize situations and factors that may cause CVP to fall short of its intended use.

CVP has been used in resuscitative efforts in patients with septic shock and as an indicator of preload in patients with hypovolemia.[1,4] It is used as an end point of resuscitation for humans in septic shock, as illustrated in the landmark study done by Rivers et al.[1] However, CVP may not be an accurate indicator of acute blood loss. In humans, acute hypovolemia due to blood loss may lead to a decrease in ventricular compliance and an increase in CVP, making CVP measurements less reliable during trauma resuscitation.[41] Although CVP generally decreases when blood volume is reduced, as illustrated in a study by Haskins et al,[42] an animal with life-threatening hypovolemia may have a normal CVP.[9,41]

CVP and other invasive cardiac measurements, such as pulmonary capillary wedge pressure, have been used to aid in the assessment of patients with heart disease.[43] A markedly increased CVP in a person with acute congestive heart failure confirms myocardial failure. However, the CVP measurement is rarely useful to guide fluid therapy or treatment in these patients.[44] In dogs, congestive heart failure is more commonly caused by valvular disease.[45] Regardless of the underlying etiology, the effect of fluid administration on patients in congestive heart failure remains a concern. Invasive monitoring, including pulmonary capillary wedge pressure (PCWP) and CVP, has been used to try to tailor this therapy. A PCWP represents the pressure in the pulmonary venous circulation, and readings over 18 mm Hg have been shown to be consistent with pulmonary edema formation.[44] PCWP represents filling pressures affecting the left side of the heart, whereas the CVP represents the right-sided filling pressures. While there may be a close relationship between CVP and PCWP in healthy individuals,[21,44] CVP measurements may not represent pulmonary venous pressure in patients with left-sided congestive heart failure.[22] This is because the ability of the right and left ventricles to receive and pump blood is different in congestive heart failure.[22] The filling pressure of the right ventricle (CVP) may not accurately reflect the effects of volume infusion on the left ventricle and the pulmonary circulation.[18] Also, the effects of therapy must also be considered. For example, it is not uncommon for human patients in heart failure receiving diuretic therapy to have a low CVP and high PCWP.[18]
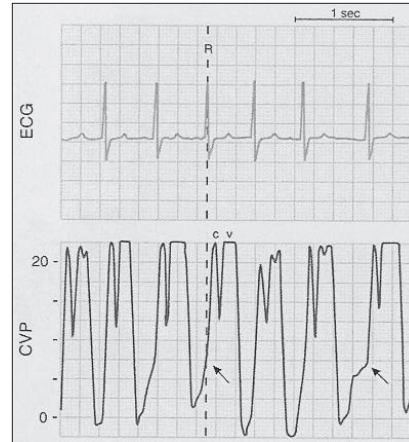
Overall, while CVP measurements should not be used as an indicator of left-sided congestive heart failure, a CVP >10 to 12 cm H_2O may be associated with the formation of pulmonary edema in people.[18,31]

During fluid therapy (i.e., during a prolonged stay in the intensive care unit), CVP may be a poor indicator of volume overload.[9] Clinical signs of volume overload may be readily apparent on physical examination long before there is a significant increase in CVP, especially in patients with left-sided heart disease.[4] Clinical signs associated with fluid overload may include serous nasal discharge, tachypnea, chemosis, dyspnea, tachycardia, subcutaneous edema, pitting edema, pulmonary crackles, excessive weight gain, shivering, nausea, vomiting, and restlessness.[46] While clinical signs of fluid overload may precede changes in CVP measurements, an upward trend of the CVP may warrant investigation into the patient's hydration status.[9]

While trends in CVP values and response to fluid boluses can be used in conjunction with other perfusion parameters to assess preload, evaluation of the CVP waveform can demonstrate indicators of other disease processes. Predictable waveform changes can occur in patients with cardiovascular abnormalities such as cardiac arrhythmias, tricuspid valve disease, and pericardial effusion.[19,28] Junctional rhythm disturbances may lead to delayed retrograde atrial depolarization. This delay may cause the right atrium to contract against a closed tricuspid valve, producing large waves called *cannon a waves*[19] (**FIGURE 10**).[10] Tricuspid stenosis may also lead to large a waves due to resistance of blood flow through the tricuspid valve causing increased atrial pressure during right atrial contraction.[28] Tricuspid regurgitation, on the other hand, may lead to giant v waves, or ventricularization, as the pressure generated during right ventricular contraction is detected at the catheter tip[28] (**FIGURE 11**).[10] In cases of cardiac tamponade, the x descent steepens as increased right atrial pressure hastens emptying of blood into the right ventricle through the tricuspid valve.[28] Interpretations of waveforms in veterinary medicine can be challenging but offer another opportunity to evaluate the patient.

Conclusion

The relationship between right atrial pressure, CVP, right ventricular pressure, right ventricular end-diastolic volume, stroke volume,

Key Points:

- CVP is a reflection of right atrial pressure, which is used as an estimate of left ventricular end-diastolic volume (preload).

- CVP is determined by the interaction of cardiac function and venous return.

- The physiologic or pathophysiologic significance of CVP should be interpreted in the light of corresponding measurements of cardiac output or a surrogate thereof.

- Isolated CVP measurements are of limited value. Instead, trends of CVP measurements over time or changes in response to a fluid challenge may provide more reliable information regarding intravascular volume status.

and cardiac output is complex and dynamic. CVP is determined by cardiac function and venous return; therefore, the physiologic and pathophysiologic significance of a given CVP cannot be interpreted without an idea of cardiac output or a surrogate thereof at the time of the measurement. Single CVP measurements have little relation to intravascular volume or cardiac output.[9] Even when used to evaluate the hemodynamic response to a fluid challenge, the CVP can be misleading, especially if the fluid challenge is administered slowly.[4,9] For clinicians who have a clear understanding of its underlying physiologic principles and limitations, CVP may provide useful information about a patient's cardiovascular status.

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CE

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1. **A high CVP reading in conjunction with a large fluctuation of the waveform synchronous with the heartbeat is most indicative of**
   a. the presence of the catheter tip in the right ventricular lumen.
   b. volume overload.
   c. the catheter tip touching the vessel wall.
   d. left-sided heart failure.
   e. a and d.

2. **Which of the following does not cause an increase in CVP?**
   a. increased abdominal pressure
   b. pleural effusion
   c. increased right ventricular compliance
   d. positive-pressure ventilation
   e. lateral recumbency

3. **Which statement about the CVP waveform is false?**
   a. The a wave represents decreased atrial pressure during right atrial contraction.
   b. The c wave represents a bulge of the tricuspid valve into the right atrium during ventricular systole.
   c. The v wave represents the phase of rapid atrial filling, during which the tricuspid valve is closed.
   d. The x descent represents reduction of right atrial pressure during ventricular ejection.
   e. The y descent represents a decrease in right atrial pressure after the tricuspid valve opens and the right ventricle fills rapidly.

4. **Because the relationship between ventricular end-diastolic volume and pressure is curvilinear and may indirectly represent cardiac output and right atrial pressure, which of the following statements is false?**
   a. After a fluid bolus, in the plateau phase of the curve, right atrial pressure (or CVP) will continue to increase, and cardiac output will no longer increase.
   b. Continued fluid administration will lead to indefinite increases in ventricular end-diastolic volume, stroke volume and, therefore, cardiac output.
   c. Ventricular end-diastolic pressure will continue to increase after continued fluid boluses even though stroke volume (and therefore cardiac output) no longer increases.
   d. The relationship between cardiac output and right atrial pressure is curvilinear.
   e. The increase in right atrial pressure (or its surrogate, CVP) seen after a fluid bolus is due to the Frank-Starling relationship of the heart.

5. **CVP is determined by the interaction between cardiac function and venous return, which are in turn influenced by all of the following except**
   a. intrathoracic pressure.
   b. cardiac output.
   c. vascular tone.
   d. right ventricular compliance.
   e. pulmonary artery occlusion pressure.

6. **CVP measurements are made in reference to atmospheric pressure but are determined by transmural pressure, which may be affected by all of the following except**
   a. positive-pressure ventilation.
   b. cardiac tamponade.
   c. blood volume.
   d. pleural effusion.
   e. intraabdominal pressure.

7. **Water manometers may overestimate CVP by**
   a. 0.5 to 5 cm H_2O.
   b. 0.5 to 5 mm Hg.
   c. 0 to 10 mm Hg.
   d. −3 to +3 cm H_2O.
   e. 5 to 10 cm H_2O.

8. **Which of the following statements is false?**
   a. Trends of CVP in response to fluid challenges are most helpful for determining preload.
   b. Isolated CVP measurements should not be used to assess blood volume.
   c. CVP should be interpreted in light of cardiac output or other perfusion parameters after a fluid bolus.
   d. The type, rate, and volume of fluid administered have little bearing on interpretation of the value of the CVP.
   e. The effect of a fluid bolus on cardiac output should be immediate.

9. **Which of the following statements is false?**
   a. CVP has consistently been shown to be closely correlated to other indicators of right ventricular volume, stroke volume, and other indicators of cardiac performance following a fluid bolus.
   b. CVP is not useful for determining whether a patient will experience fluid overload from left-sided heart disease.
   c. Patients show other signs of fluid overload before they show consistent elevations in CVP.
   d. A combination of an increase in CVP and increase in cardiac output (or improvement in other perfusion parameters) indicates that the patient is fluid responsive.
   e. CVP measurements may be affected by cardiac arrhythmias, hypovolemia, changes in intrathoracic pressure, and patient position.

10. **A cannon a wave represents**
    a. increased atrial pressure from increased intrathoracic pressure.
    b. increased atrial pressure from increased blood volume.
    c. increased atrial pressure from atrial contraction against a closed tricuspid valve.
    d. decreased atrial pressure from decreased blood volume.
    e. decreased atrial pressure from tricuspid regurgitation.