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Central venous pressure monitoring

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Clinical management of the critically ill animal usually requires regular assessments of the adequacy of intravascular blood volume and cardiac preload. Hemodynamic instability may develop in many different ways. Excessive vomiting, diarrhea, polyuria, hemorrhage, or body cavity effusion can result in hypovolemia. Sepsis or systemic inflammatory response syndrome may alter vascular volume and tone. Central venous pressure (CVP) measurement can provide useful information in these and other situations. It represents the direct measurement of venous blood pressure from a catheter inserted into the cranial vena cava or, less commonly, into the caudal vena cava or the right atrium. When measured from the vena cavae, CVP is considered to be a close approximation of right atrial pressure,[1] which in turn is a determinant of right ventricular preload. As a result, CVP measurement is widely used as an index of circulating blood volume to help guide fluid resuscitation and diuretic therapy in veterinary patients.[2–5]

Determinants of central venous pressure

Central venous pressure is determined primarily by the interrelationship between venous return and right heart function.[6] In turn, venous return is affected by venous tone, venous wall compliance, and circulating blood volume, whereas right-sided cardiac output is determined by heart rate, preload, afterload, and contractility. Therefore any normal physiological process, disease, or medical intervention that alters one of these factors may affect CVP. This includes changes in sympathetic tone that occur during stress and illness, structural and functional diseases of the heart, and vasoactive drugs such as vasopressors, sedatives, and general anesthetics.

While these factors affect CVP due to their influence on cardiovascular function or blood volume, in some cases, extravascular forces, such as increases in intrathoracic or intra-abdominal pressure, can also change the CVP.[7,8] High pressure within the thoracic cavity can develop secondary to a number of conditions, including effusion, presence of a space-occupying mass, a forced expiratory respiratory pattern, or positive end-expiratory pressure (PEEP) during mechanical ventilation. A sufficient rise in intrathoracic pressure will compress the vena cava and right atrium and raise the CVP. Similarly, elevations in intra-abdominal pressure can be seen with ascites, acute abdominal syndrome, neoplasia, active abdominal expiratory effort, and other conditions. When there is significant disease in the thoracic or abdominal cavities, it is important for the clinician to be aware of their potential influence on the CVP. In general, when interpreting CVP measurements to help with clinical decision-making, it is crucial to remember that many things other than circulating blood volume affect the CVP.

Indications for the measurement of central venous pressure

Monitoring CVP can help to guide fluid therapy in animals with abnormalities in circulating blood volume or abnormal right heart function. Central venous pressure cannot be used to make inferences about left ventricular preload or function, however.[9] Specific indications for CVP measurement include the presence of

Advanced Monitoring and Procedures for Small Animal Emergency and Critical Care, First Edition. Edited by Jamie M. Burkitt Creedon, Harold Davis.

persistent hypotension despite fluid resuscitation, vasopressor therapy, extensive third space losses, oliguria or anuria, hemorrhage, trauma, sepsis, burns, and heart failure. Patients undergoing urgent or emergent surgical procedures, and those with multiple medical problems, can be predisposed to developing cardiovascular instability while anesthetized. In these animals it may be practical to insert a central venous catheter and begin monitoring preemptively to allow earlier detection of abnormalities in the perioperative period.

Risks

Central venous catheter use carries small but real risks of thrombosis, thromboembolism, carotid arterial puncture, infection, phlebitis, bleeding, and vascular erosion. Cardiac arrhythmias, or rarely cardiac wall perforation, can result if the catheter tip is accidentally advanced into the right atrium or right ventricle. There are fewer problems associated with CVP measurement itself. The main risk is accidental introduction of air or bacteria into the venous system, particularly if poor technique is practiced. The cranial vena cava is a low-pressure system, and opening the system to the atmosphere may result in air embolism formation. Fortunately, this is a rare occurrence with intermittent CVP measurement technique and even less common with continuous technique, as the system is closed to the outside environment. Additionally, several straightforward precautions will reduce the incidence of complications. These include following proper hand hygiene measures prior to handling the jugular catheter or CVP system, careful inspection of the tubing and transducer to assess for defects, the use of Luer lock connectors such as Luer-Lok (Becton, Dickinson and Company, Franklin Lakes, NJ), and periodic verification of the tightness of these connections, particularly in mobile patients.

Normal value

The most commonly used reference range for central venous pressure is around 0—5 cm H_2O,[3,10,11] although values of up to 10 cm H_2O can be normal.[2,12] The wide reference range reflects the inherent variability in normal resting CVP due to the impact of many factors on venous pressure, including blood volume, venous tone, and cardiac function.

The units of CVP measurement are millimeters of mercury (mm Hg) or centimeters of water (cm H_2O), depending upon the method of measurement. To convert from mm Hg to cm H_2O, multiply by a factor of 1.36:

$$\text{CVP in cm } H_2O = \text{CVP in mm Hg} \times 1.36 \quad (11.1)$$

Measurement technique

Central venous pressure is typically measured from a specialized catheter that is inserted into the external jugular vein and advanced into the cranial vena cava or, less commonly, into the right atrium. Right atrial pressure can also be measured from the proximal lumen of a pulmonary artery catheter. Pressure measurements from the vena cava and the right atrium are generally considered interchangeable at rest,[13,14] although this is untrue if there exists an obstruction between these two sites, and their responses are not identical following fluid bolus or vasopressor therapy.[15] Several alternative techniques for obtaining the CVP have been described in animals, including catheter insertion into the omobrachial vein in dogs[16] with subsequent passage into the cranial vena cava, as well as placement into the femoral vein and caudal vena cava in dogs[17] and cats.[18] In human patients, peripheral venous pressure has also been used as an estimate of CVP due to the similarities between centrally and peripherally measured venous pressures.[19,20] Unfortunately, this technique has shown poor correlation in dogs and cats.[21]

Catheter selection and placement

Either single-lumen or multilumen vascular catheters can be used and should be of sufficient length to reach the thoracic vena cava from the point of insertion. Insertion of a multilumen catheter has the added advantage of allowing continued infusion of intravenous fluids and drugs through the proximal ports as the CVP is measured simultaneously from the catheter's distal port. The rate of fluid administration has been shown to have no affect on the CVP in humans, even during pressurized saline infusions of up to 9120 mL/hour via a double-lumen catheter and up to a combined rate of 14,340 mL/hour through the proximal ports of a triple-lumen catheter.[22] These results are likely similar in veterinary patients although similar experiments have not yet been published.

Prior to catheter placement, it is important to predetermine an appropriate depth of insertion. Before approaching the jugular vein, a good estimate of depth can be obtained by measuring the distance between the insertion site and the caudal aspect of the shoulder, which should position the tip within the cranial vena cava. Following placement, the catheter's position can be verified by radiography and by visualization of a characteristic central venous pressure waveform (as discussed in the Waveform Analysis section in this chapter). Radiographic confirmation is particularly important when alternative catheter insertion sites are used, such as the saphenous vein, as premeasurement is more dif-

ficult to perform with accuracy in such cases. Catheters advanced into the right atrium can occasionally generate arrhythmias and should be backed out slightly. Accidental advancement of the catheter tip into the ventricle should be avoided. However, if it occurs, it is not difficult to recognize once a pressure measurement is obtained, as peak right ventricular pressures approach 20–30 mm Hg in the normal animal.[2] They will be visible as extreme fluctuations of the fluid column of the water manometer (for intermittent CVP measurement) or of the displayed waveform on the electronic monitor (for continuous CVP measurement).

General principles

The two methods of obtaining the CVP are intermittent measurement and continuous measurement. In both methods, the catheter is connected via a fluid-filled tubing system to a pressure-measuring device that displays the venous pressure. Intermittent measurement involves connecting the catheter to a water manometer, infusing a predetermined volume of saline, and allowing the fluid level to equilibrate with the patient's central venous pressure. The height of the fluid column within the manometer is recorded in centimeters of water pressure (cm H_2O). Continuous CVP measurement is obtained by linking the catheter via noncompliant, fluid-filled tubing to a pressure transducer that converts the venous pressure wave to an electrical signal. The venous pressure waveform is displayed continuously and in real time on a monitor. The average venous pressure in millimeters of mercury (mm Hg) is also shown.

As is commonly the case where more than one method of measurement is available, there are advantages and disadvantages to each technique. The equipment needed to perform intermittent CVP monitoring is simple and inexpensive. However, repeated measurements are relatively time-consuming to obtain. In contrast, once the system for continuous CVP monitoring is assembled, moment-to-moment changes in CVP are displayed on the monitor without the need for additional intervention. The ability to view CVP continuously is particularly useful for unstable patients in the intensive care unit and in the operating room. Frequent measurements also permit better and more timely determination of a patient's response to therapy. However, a higher level of technical knowledge and skill is needed to set up and troubleshoot continuous CVP monitoring and it also requires purchase of a specialized monitor, which can be expensive. When available, refurbished and used monitors may represent a more economical solution for many hospitals.

Zeroing and leveling

To obtain reliable pressure measurements, two principles are important. First, atmospheric pressure, which measures approximately 760 mm Hg at sea level, is used as the standard reference point to which CVP is compared. Atmospheric pressure is created by the weight of air, which presses down on the body and everything within it, including the central veins. It also pushes on the transducer and the manometer fluid column. Since it exerts the same magnitude of pressure on each object, for simplicity it is cancelled out and atmospheric pressure is considered 0 cm H_2O (0 mm Hg) for purposes of CVP measurement. This allows a CVP of 770 mm Hg to be read as 10 mm Hg and eliminates the need to adjust CVP measurements for fluctuations in barometric pressure.[23] The process of correcting for atmospheric pressure is called **zeroing**. Pressure transducer systems have an integrated stopcock and port that can be opened to the atmosphere to calibrate, or *zero*, the transducer.

The second principle is the importance of aligning the transducer system (or manometer) with the vascular structure containing the pressure of interest, also known as the **zero reference point**, in order for the measurements to be accurate. In the case of CVP, the zero reference point is the center of the right atrium. The stopcock (for continuous CVP measurement) or the 0 cm H_2O mark on the manometer (for intermittent measurement) must be positioned or **leveled** on the same horizontal plane as the patient's right atrium during zeroing and thereafter during measurement, as demonstrated in Figure 11.1. See Chapter 8, Fluid-Filled Hemodynamic Monitoring Systems, for more information regarding zeroing and leveling transducers.

Due to species- and breed-related variability in thoracic conformation, there is no completely foolproof method of determining where the right atrium lies. However, as a general rule of thumb, the sternum is a good approximation for a dog or cat in lateral recumbency. For an animal in sternal recumbency, draw an imaginary vertical line at the caudal aspect of the shoulder that extends from the top of the dorsal spinous process to the sternum. The right atrium lies at a point that is roughly 40% of the height of this line.

Proper determination of the zero reference point is crucial, because *failure to level the transducer (or manometer) relative with it will result in an erroneous central venous pressure reading*. It will be falsely high if the transducer is resting below the right atrium and falsely low if the transducer is above the right atrium; if the error is not detected and the resulting measurements are used as the basis for fluid therapy decisions, harm to the patient could result. For this reason, the height of the

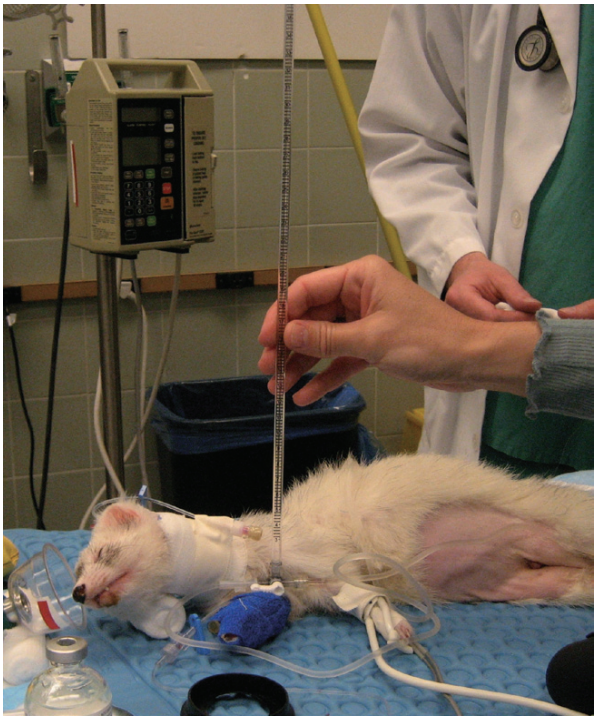

Figure 11.1 Leveling of the manometer with the zero reference point. Though this ferret's heart is far caudal of the manometer, note that the zero value must only be on the same horizontal plane as the patient's right atrium in order for measurements to be accurate.

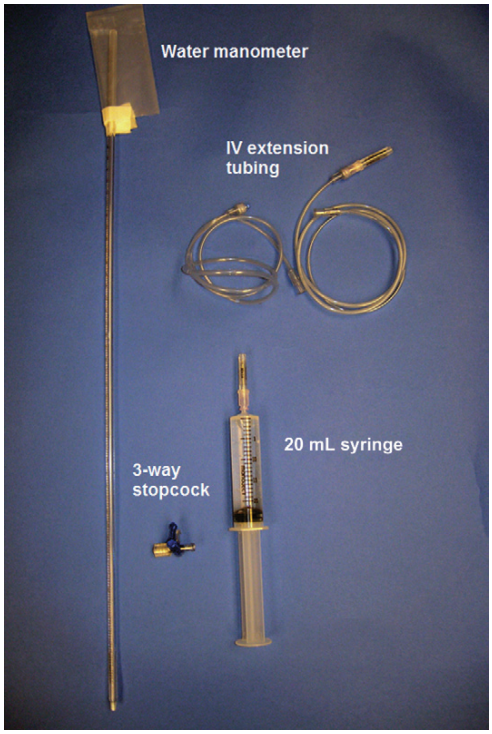

Figure 11.2 Equipment required for intermittent central venous pressure measurement.

transducer and stopcock system should always be evaluated prior to each pressure reading. Their position may need to be adjusted depending on whether the animal is recumbent, sitting, or standing. Gentle restraint may be needed with mobile patients to obtain accurate CVP readings. The transducer should also be periodically rezeroed—always at the level of the right atrium—due to the potential for drift.[24] Additionally, whenever possible, measurements should be taken by the same person to avoid inconsistent technique. The attending veterinarian should be notified if the CVP measurements are showing an upward or downward trend, particularly if they fall outside of expected or target values.

Intermittent central venous pressure measurement

Prior to initiating CVP measurement, it is important to verify the patient has a correctly positioned central venous catheter, as described earlier in this chapter. Two types of water manometers can be used for intermittent CVP measurement. The best one is a rigid, narrow, cylindrical tube made of glass or plastic specifically manufactured for this purpose that has centimeter markings along its length. A simpler homemade alternative consists of a short section of IV fluid extension tubing that can be affixed to a ruler. Either standard fluid tubing or noncompliant tubing manufactured specifically for blood pressure monitoring can be used. The equipment required to perform intermittent CVP measurement is shown in Figure 11.2.

To ensure accuracy, the patient should rest in the same position for each measurement period and this position should be recorded on the patient's treatment sheet for future reference. Both lateral and sternal recumbencies are acceptable. It may be more difficult to obtain accurate readings from an animal that is sitting or standing. Whenever possible, use the same position for subsequent measurements. In some situations, it will be impractical or unsafe to position the patient in a certain way. If this is the case, it is important to adjust the height of the manometer so that the 0 cm H_2O mark is level with the zero reference point (the right atrium) prior to recording the CVP. Alternatively, as CVP can occasionally fall below zero, it can be helpful to align the 10 cm H_2O mark with the zero reference point to facili-

tate measurement of negative pressures. When this positioning is used, one must subtract 10 cm H_2O from the height of the fluid column in the manometer to obtain the true CVP measurement.

The CVP is read at the bottom of the meniscus in the manometer (or at the center of the floating ball in the manometer, if present). Patient heartbeat or respiration may cause millimeter fluctuations in the meniscal level. In a spontaneously breathing animal, the CVP reading will decrease during inspiration and increase during expiration. The pressure reading should be obtained at end-expiration if the patient is breathing normally. If there is pronounced expiratory abdominal effort, the measurement should be obtained at the beginning of the expiratory phase, prior to the onset of active abdominal effort.[1,6] Detailed instructions on performing intermittent CVP measurement are available in Protocol 11.1.

The time interval between measurements will depend on the patient's cardiovascular status as well as personnel availability, but a reasonable place to start is to space readings 1–4 hours apart. If more frequent measurements are desired, continuous CVP measurement should be considered.

Protocol 11.1 Intermittent central venous pressure measurement

Items Required

- Indwelling central venous catheter (inserted to the proper depth)
- Disposable water manometer (calibration in centimeters), or a centimeter ruler and extension tubing
- 0.9% NaCl
- Three-way stopcock with locking fittings
- 20-mL syringe
- Noncompliant tubing or standard fluid extension tubing
- Heparinized saline flush
- One assistant

Procedure

1. Collect the necessary supplies.
2. Perform hand hygiene and don examination gloves.
3. The patient may lie in lateral or sternal recumbency. This position should be recorded on the patient treatment sheet for future reference.
4. If applicable and safe to do so, have the patient rest in the same position that was recorded in the treatment sheet for prior CVP measurements.
5. Flush the patient's central venous catheter with heparinized saline solution to ensure patency.
6. Assemble the CVP monitoring setup. First orient the manometer vertically. Then connect the three-way stopcock to the manometer, to the saline-filled syringe, and to the fluid tubing.
7. Orient the three-way stopcock valve so that it is closed to the manometer and open to the tubing and fluid-filled syringe. Then, prime (fill) the stopcock and fluid tubing with 0.9% NaCl from the syringe. Connect the fluid tubing to the central venous catheter. If a multilumen catheter is being used, connect the tubing to the central lumen.
8. Orient the stopcock valve so that it is closed to the patient's central venous catheter and open between the manometer and the fluid-filled syringe. Using the 20-mL syringe, fill the manometer with 0.9% NaCl to a level that is approximately 10–20 cm H_2O greater than the patient's expected CVP. Do not allow the manometer to overflow while filling.
9. The manometer may be attached to an IV pole to facilitate height adjustments, held vertically in the operator's hand, or taped to the wall of the cage. Locate the 0 cm H_2O mark and position the manometer so that it is level with the zero reference point (the patient's right atrium).
10. Close the stopcock valve toward the syringe, which will open a fluid column between the manometer and the patient. The fluid level in the manometer will initially fall as it flows into the patient and then stabilize as the fluid level equilibrates with the CVP.
11. The operator should ensure consistent readings by performing three or more consecutive measurements and calculating an average. Record the average pressure and patient position on the patient's treatment sheet.
12. When the measurements have been completed, turn the stopcock off to the manometer and disconnect the pressure tubing from the central venous catheter. If intravenous fluid therapy will not be immediately resumed, flush the central venous catheter with heparinized saline.

Continuous central venous pressure measurement

The list of supplies and equipment needed for continuous CVP measurement is more extensive than it is for intermittent measurement. To better familiarize the reader with the materials and monitoring technique, additional details are provided here.

Pressure transducer kits come presterilized and typically consist of a disposable pressure transducer, three-way stopcock with zeroing port cap, and a flush mechanism (see Fig. 9.1). Some also include noncompliant ("high-pressure") tubing that is needed to join the pressure transducer to the patient's central venous catheter. Additional tubing can be added between the transducer and the patient if more length is needed. Standard fluid extension tubing is soft and compliant, whereas noncompliant tubing made for blood pressure measurements is flexible but nondistensible. In theory, excessively long or compliant tubing could dampen transmission of the pressure waveform, resulting in waveform distortion.[25,26] Due to the low vascular pressures of the venous system, damping from overly compliant tubing is less of a concern with CVP measurement than it is with direct arterial blood pressure monitoring.[26] But optimization of the CVP waveform will be achieved with noncompliant tubing specifically manufactured for blood pressure monitoring. The reader is referred to Chapter 8, Fluid-Filled Hemodynamic Monitoring Systems, for more details regarding tubing characteristics.

The last element of the continuous monitoring setup is assembly of the flush system, which is pressurized and provides a simple method of flushing the catheter by pulling the rapid flush valve (usually referred to as a *pigtail* on the transducer). The flush solution consists of heparinized saline and it is prepared by adding unfractionated heparin to a bag of 0.9% NaCl to a final concentration of 1 U/mL.

Prior to beginning measurement, the transducer must be zeroed at the proper level by exposing it to atmospheric pressure in approximately the same horizontal plane as the right atrium (see Intermittent Measurement instructions for more details). The fluid-filled system is zeroed and leveled by turning the stopcock lever off toward the patient, removing (and saving) the zeroing port cap, and setting the monitor to zero. The reader should consult the reference manual for his or her specific monitor for additional details. Once this has been done, the monitor will display 0 mm Hg and the waveform tracing should overlap zero on the displayed scale. If the transducer does not zero, the transducer is the most likely culprit, and it should be replaced and the procedure reattempted. After the zeroing step, the zeroing port cap is replaced and the stopcock is turned so it points off toward the port cap to create a continuous fluid column between the pressure transducer and the patient's central vein. Central venous pressure measurement may then begin.

As with intermittent CVP measurement, it is important to ensure the transducer is at the level of the zero reference point prior to zeroing or obtaining a reading. This is best achieved by having the patient lie in the same position for each measurement period. Specific instructions for assembling the continuous CVP measurement system are included in Protocol 11.2. Common issues and solutions that may be encountered during continuous CVP monitoring are summarized in Table 11.1.

Maintenance of the continuous CVP system

For the patient undergoing continuous CVP monitoring, certain maintenance procedures should be included in the patient's treatment orders, listed in Box 11.1. The patient can be disconnected from and reconnected to the CVP measurement system without the need to rezero the transducer as long as the disconnection occurs between the transducer and the central venous catheter. However, if the transducer cable is disconnected from the monitor, it will be necessary to rezero the transducer prior to obtaining a CVP reading.

CVP interpretation

Central venous pressure should never be used as the sole monitoring parameter to determine the adequacy of circulating blood volume. It must always be evaluated in conjunction with the patient's history, signalment, and physical examination, and ideally with knowledge of the animal's cardiac and renal function. Laboratory data

Box 11.1 Maintenance of the continuous CVP system

- The transducer and catheter system should be periodically flushed by pulling on the fast flush device. This should be performed at least once every 4 hours.
- The transducer should be rezeroed no less frequently than every 12 hours due to the potential for drift.
- Change the flush solution and tubing every 48 hours.
- Ensure there are no air bubbles in the fluid line at any time.
- Periodically inspect and reinflate the pressure bag to 300 mm Hg as necessary, and verify the heparinized saline bag is not empty.

Protocol 11.2 Continuous central venous pressure measurement

Items Required
- Indwelling central venous catheter inserted to the proper depth
- Pressure transducer kit
- Bag of 0.9% NaCl heparinized to a concentration of 1 U/mL
- Standard fluid administration set
- Pressure bag of appropriate size for heparinized saline bag
- Ideally, noncompliant fluid tubing; alternately, standard fluid extension tubing
- Electronic blood pressure monitor and its associated transducer cable

Procedure
1. Collect the necessary supplies.
2. Perform hand hygiene and don examination gloves.
3. The patient may lie in lateral or sternal recumbency. This position should be recorded on the patient treatment sheet for future reference.
4. Flush the patient's central venous catheter with heparinized saline solution to ensure patency.
5. Assemble the heparinized saline flush system by spiking the heparinized saline fluid bag with the standard fluid administration set and flushing fluid through the tubing. Clamp the tubing and cap the set's open end.
6. Place the pressure bag over the bag of heparinized saline, hang it on an IV pole placed next to the patient, and inflate the pressure bag to 300 mm Hg.
7. Assemble the transducer system by connecting it to the noncompliant tubing, to the assembled heparinized saline flush system, and to the transducer cable and electronic monitor, as shown in Figure 9.1.
8. Prime (fill) the transducer and noncompliant tubing system with heparinized saline by pulling the transducer's fast flush valve (pigtail).
9. Connect the pressure tubing to the central venous catheter. If using a multilumen catheter, the CVP measurement should be obtained from the most distal lumen, reserving the other lumens for IV fluid therapy, drug administration, or blood withdrawal. It is not necessary to discontinue IV fluid or drug administration through the other ports during CVP measurement.
10. Position the height of the transducer and stopcock system so that the stopcock is aligned at the same height as the zero reference point (the right atrium). The transducer may be attached to an IV pole, to the cage door, or taped to a stable support that is resting on the floor of the cage.
11. Flush the catheter by pulling on the pigtail and wait for the pressure to equilibrate.
12. The system must be calibrated (zeroed) before any measurements can be interpreted. To perform zero-calibration, with the stopcock at the height of the right atrium, turn the stopcock off toward the patient. Remove the zeroing port cap on the stopcock to open it to the atmosphere. Select the zeroing function on the display monitor and wait for it to read 0 mm Hg. Replace the zeroing port cap and turn the stopcock toward the zero port, which will allow the pressure to equilibrate between the patient's vena cava and the transducer.
13. Once the pressure has stabilized, record the CVP measurement.
14. For subsequent measurements, verify the transducer height is correctly positioned at the zero reference level before recording the CVP measurement.

and additional markers of hemodynamic status, such as arterial blood pressure and urine output, should also be factored into the clinical assessment.

Due to the number of potential factors that can influence CVP, an isolated value is difficult to interpret and is of minimal benefit. However, serial measurements over time may document trends in venous blood pressure that can provide useful information to assist in the assessment of circulating volume status. Apart from its usefulness as a diagnostic tool when hypovolemia is suspected, CVP measurement can also be used to monitor the effectiveness of fluid therapy to treat low circulating blood volume. However, in a similar manner, its success or failure should be determined by concurrently evaluating a combination of other clinical markers.

In hemodynamically unstable patients, the primary objective of fluid therapy is to optimize right ventricular preload in an effort to improve cardiac output and tissue perfusion. The rationale for performing CVP measurement is its ability to serve as an estimate of right atrial pressure, which is a major determinant

Table 11.1 Troubleshooting tips for continuous CVP measurement

Problem	Possible Cause
Pressure is displayed as a flat line rather than a waveform.	Complete occlusion of the catheter, stopcock, or fluid line (displayed pressure will be far above the normal reference range).
	Partial occlusion of the catheter, stopcock, or fluid line.
	Patient is small (cats and small dogs occasionally lack a visible waveform although the mean pressure can still be used for trending purposes).
	Air bubble or leak in the system.
No pressure is displayed on the monitor.	Monitor display settings are incorrect.
	Transducer was not zeroed.
	Transducer cable is broken or is not plugged into the monitor.
Pressure reading is higher than expected.	Intrathoracic or intra-abdominal pressure is significantly increased.
	Central venous catheter is clamped off or occluded.
	Transducer is below the level of the right atrium.
	Transducer is defective.
Pressure reading is lower than expected.	Transducer is above the level of the right atrium.
	Transducer is defective.
Waveform is "noisy."	Patient movement.
	Panting.
	Catheter tip is within the heart.
	Arrhythmia.
Sudden change in pressure.	Hemodynamic instability.
	Transducer position relative to the zero reference point (right atrium) has changed.
System does not flush.	Stopcock position is incorrect.
	Pressure bag is not sufficiently inflated.
	Heparinized saline bag is empty.
	Heparinized saline administration line is clamped off or occluded.
	Central venous catheter is clamped off or occluded.

of right ventricular end-diastolic pressure. Right atrial and right ventricular pressure are equal when the tricuspid valve is open and pressures have equilibrated at the end of ventricular diastole. Right ventricular end-diastolic pressure is in turn related to right ventricular end-diastolic volume, which determines end-diastolic myocardial wall stretch, or **preload**.

While the initial temptation would be to assume that low values of CVP correspond to hypovolemia and high values indicate volume overload, in reality, the association between CVP and preload is not always straightforward. Critics note that isolated values of CVP do not correlate well with intravascular blood volume, nor can CVP be used to predict stroke volume or cardiac output following a fluid challenge.[27] Despite these limitations, its proponents argue that CVP nevertheless provides useful information about preload and right-sided heart function and that these criticisms reflect misunderstanding and misuse of this monitoring tool.[14] A review of the physiological principles behind venous return and venous pressure is useful here to highlight their reasoning.

Venous return describes the flow of blood from the systemic circulation back to the heart. Proper flow depends on the maintenance of an adequate pressure gradient, often referred to as the **driving pressure**, between the peripheral and central venous vasculatures. Driving pressure here is small. The pressure in the peripheral venous circulation averages only 5–10 mm Hg greater than the pressure within the central veins, and homeostatic adjustments ensure continued return blood flow as variables change.[7]

The dynamic properties of the venous system that allow it to regulate venous return also govern its other role, which is to serve as a blood reservoir. Veins contain approximately 65% of the systemic blood volume.[28] A major portion of that blood is contained within the splanchnic veins, which function as capacitance vessels capable of significant adjustments in wall compliance to accommodate changes in blood volume. When effective

circulating volume is low, constriction of the splanchnic veins increases the circulating pool of blood to help support adequate venous return.[7] Central venous pressure may change minimally during this time despite the recruitment of additional volume, and clinically, the patient may appear to be coping quite well. However, once the blood reservoir has been depleted and other compensatory mechanisms have been exhausted, the patient will decompensate suddenly and CVP will fall.

The complex relationship between CVP and circulating blood volume may explain why a CVP within the normal reference range cannot distinguish the normovolemic patient from one with compensated hypovolemia or hypervolemia, due to homeostatic mechanisms that attempt to maintain an adequate pressure gradient for venous return. A severely elevated CVP may be due to normovolemia in the presence of severe cardiac dysfunction, or hypervolemia with adequate cardiac performance.[14] The complexity of these interactions may explain why studies have consistently failed to find a threshold CVP pressure below which fluid loading will always improve cardiac output.[27,29]

Despite these limitations, several generalizations about CVP interpretation can be made that ensure its continued usefulness in the ICU. First, CVP should be regarded as a probable, rather than an absolute, indicator of volume status. In other words, in the presence of normal cardiac function, patients with a low CVP are more *likely* to respond to volume than patients with a normal or high CVP. In human patients with severe circulatory dysfunction, a CVP less than 5 mm Hg has been shown to be an excellent positive predictor of fluid responsiveness.[30] In contrast, those with a CVP greater than 10–12 mm Hg are unlikely to benefit from a fluid bolus, although some still can.[1,14] Those that do respond may have a condition such as elevated intrathoracic or intra-abdominal pressure that is causing the CVP to overestimate the true transmural pressure.[1,8]

The second generalization that can be made is that rising or falling trends are clinically meaningful. A progressive drop in CVP should alert the clinician to the possibility of ongoing and excessive internal or external fluid losses, particularly if supported by the presence of other markers of hypoperfusion. In contrast, a rising CVP with concurrent evidence of worsening tissue perfusion may indicate declining cardiac function as the cause, and may suggest that additional fluid loading is unwise.[1] With the latter scenario, additional caution is warranted if hypoalbuminemia or vasculitis is present, as either of these conditions increases the risk of edema formation with intravenous fluid therapy. A summary of the possible clinical interpretations of rising or falling trends in CVP is provided in Table 11.2.

Potential sources of interpretation errors

As discussed earlier, extravascular forces, such as significant elevations in the pressure within the thoracic or abdominal cavities, can raise CVP. An increase in CVP of approximately 3 mm Hg was seen at PEEP levels of 10 cm H_2O in humans[31] and 15 cm H_2O in pigs.[32] However, in the absence of PEEP, an alteration of tidal volume alone (8 mL/kg versus 16 mL/kg) did not result in a similar effect.[31] Large elevations in intra-abdominal pressure, secondary to acute abdominal syndromes, ascites, or a forced expiratory respiratory pattern, can also lead to changes in the CVP due to transmission of the increased abdominal pressure across the diaphragm to the thoracic cavity.[8,29]

It is important to recognize that the higher CVP generated by an elevation in intrathoracic or intra-abdominal pressure *does not necessarily result in changes in driving pressure and venous return*. This is because the physiological variable that ultimately governs distension of the central veins is **transmural pressure**, not CVP.[1,6]

Table 11.2 Clinical interpretation of CVP measurement trend[21]

CVP Trend	Possible Cause
Low or falling	Shock Vasodilation
Normal	Normovolemia Compensated hypovolemia Compensated hypervolemia
High or rising	Volume overload Vasoconstriction or systemic hypertension Right-sided heart disease • Tricuspid regurgitation • Tricuspid stenosis Pericardial disease • Pericardial effusion • Constrictive pericarditis Vena caval obstruction Pulmonary disease • Pulmonary hypertension • Pulmonary thromboembolism Increased intrathoracic pressure • Pleural effusion • Intrathoracic mass • PEEP • Positive-pressure ventilation in the presence of hypovolemia • Pneumothorax Increased intra-abdominal pressure Occlusion of the catheter, fluid line, or stopcock

The concept of transmural pressure is best understood by recognizing that venous distension depends not only on the pressure exerted on the vascular wall from the inside (CVP), but also on the pressure exerted from the outside, and this is pleural pressure, not atmospheric pressure. The net difference (intravascular pressure minus extravascular pressure) is called transmural pressure. Due to the inherent difficulties in obtaining pleural or pericardial pressures in the clinical setting, transmural pressure is usually not directly determined. Fortunately, it corresponds fairly closely to CVP under most conditions. However, this relationship can unravel during certain situations, notably with increases in intrathoracic or intra-abdominal pressure.[7,8] In the PEEP example described above, the increased alveolar pressure generated by PEEP is transmitted to the heart and intrathoracic vessels and results in an elevation in CVP. However, PEEP is also transmitted to a similar extent to the pleural and pericardial spaces. As a result, central venous transmural pressure changes minimally.

However, as we have already mentioned, venous return depends ultimately on driving pressure, which is the difference between peripheral and central venous pressures. Driving pressure was previously defined as the difference between peripheral and central venous pressures. However, it is more accurately defined as the difference between peripheral and central *transmural* venous pressures. So in this example, although there has been an absolute increase in measured CVP, transmural central and peripheral venous pressures have remained steady. Therefore there is no change in the rate of return of blood to the heart.

Performing a fluid challenge

When CVP is extremely low or falling, the index of suspicion for hypovolemia should be high. Usually, an evaluation of other clinical markers will support an assessment of low circulating blood volume, and fluid resuscitation can be started immediately. However, on occasion, these findings will be unclear or contradictory; when this occurs, a fluid challenge is the classic method of verifying fluid responsiveness.

The idea is to give a small test volume as a rapid bolus and to monitor for an improvement in clinical perfusion parameters such as patient alertness, pulse quality, pulse rate, mucous membrane color, and capillary refill time. Faster administration reduces the volume needed to achieve an effect. If a beneficial response is seen following the volume challenge, additional fluid is given until the desired endpoint is reached. Under ideal monitoring circumstances this endpoint would be an increase in cardiac output. However, as cardiac output is difficult to measure without cardiac catheterization or other specialized techniques, several indirect indices are more commonly used, such as improved systemic arterial blood pressure, lower blood lactate concentration, increased urine production, and higher central venous oxygen saturation, as well as improvement in physical examination findings. See Chapter 15, Monitoring Tissue Perfusion: Clinicopathologic Aids and Advanced Techniques, for more information about many of these indirect perfusion indices.

A fluid challenge is performed by rapidly infusing a small volume of crystalloid or colloid using a pressure bag or fluid pump. Useful crystalloid test volumes are 15 mL/kg in the dog, or 5 mL/kg in the cat. If a colloid is used, 5 mL/kg in the dog or 2.5 mL/kg in the cat are reasonable. The fluid bolus is given over 10–15 minutes and the animal is monitored for signs of improved perfusion and CVP.

The classic response to a fluid challenge in the euvolemic animal is a rise in CVP of 2–4 cm H_2O, followed by a rapid return to the original value within 15 min.[33] However, if the starting CVP is low and it rises minimally or rapidly returns to baseline (within 5–15 minutes) following a fluid challenge, hypovolemia is likely,[33,34] particularly when corroborated by other findings as mentioned previously. The fall in CVP is due to the redistribution of fluid from the intravascular to the interstitial space, stress-induced relaxation of venous tone, and pooling of blood within the splanchnic vascular bed.[35] In contrast, a persistent, marked elevation in CVP following a fluid challenge, or a prolonged return to baseline (greater than 30 minutes) may support volume overload, decreased cardiac performance, or restrictive pericardial disease such as tamponade.[33]

It should be noted that these guidelines reflect general trends, not absolute rules. A low CVP will not always indicate that a patient has inadequate blood volume, just as a high CVP does not necessarily signify fluid excess or cardiac dysfunction.[14] Normovolemic animals may show a rise in CVP following a test bolus, even though they do not actually require fluid. Therefore, there is no substitute for careful patient assessment and clinical judgment when using CVP measurement to help guide fluid therapy.

The normal CVP waveform

Blood pressure in the central veins is pulsatile as a result of pressure changes in the right heart during the cardiac cycle. The baseline pressure also fluctuates from changes in intrathoracic pressure generated by the phases of respiration. Both intermittent and continuous CVP mea-

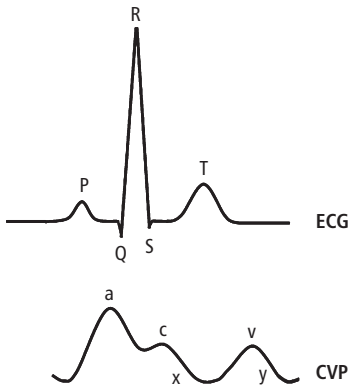

Figure 11.3 The relationship between the waves and descents of the CVP waveform and the electrocardiogram. Under normal circumstances, the mean of the *a* wave provides the best estimate of CVP because it corresponds to the venous pressure at the end of diastole. Ventricular systole begins immediately following the appearance of the QRS complex on the ECG.

surement techniques provide a mean CVP, although in the case of intermittent measurement, small pulsations are often evident in the fluid column.[33]

In a similar manner, the CVP reported during continuous measurement represents a mathematical average of this variable pressure. The pressure waveform is displayed on the monitor and classically consists of three "waves" (positive deflections from baseline: the *a* wave, *c* wave, and *v* wave) and two descents (negative deflections: the *x* descent and the *y* descent), which correspond to specific right atrial and right ventricular cardiac events (see Fig. 11.3).[36] Not all waves and descents will be evident in all CVP tracings and considerable individual variability in waveform appearance can be seen. Waveforms can be small or impossible to discern in cats and small dogs, and may be absent if the catheter lumen becomes partially occluded (such as by a blood clot).

The *a wave* is generated by atrial contraction and appears shortly after the P wave on an electrocardiogram. The *a* wave is followed by the *x descent,* which reflects a decrease in right atrial pressure caused by atrial relaxation. The *c wave* is sometimes visible as a secondary peak following the *a* wave and is caused by bowing of the tricuspid valve into the atrium during early right ventricular systole. As atrial diastolic filling proceeds, the *v wave* is created and it is generated soon after the T wave on an electrocardiogram. When ventricular systole ends and the ventricle relaxes, atrial pressure exceeds ventricular pressure, the tricuspid valve opens, and blood flows from the right atrium into the ventricle. Atrial emptying leads to a decrease in atrial pressure, thus producing the *y descent*. The *a* wave is usually larger than or similar in size to the *v* wave.[36]

Determining the CVP from the normal waveform

During continuous CVP measurement, the monitor displays a single pressure reading that represents an average pressure measurement over time. The mean CVP is often sufficient for clinical assessment in patients lacking significant primary cardiovascular or respiratory disease. However, changes in respiratory pattern, cardiac arrhythmias, and diseases that alter filling, emptying, or compliance of the right heart can lead to an inaccurate estimation of ventricular end-diastolic pressure (preload), resulting in errors in clinical interpretation. When any of these problems are evident, it is important to determine the CVP directly from the printed venous pressure tracing to ensure accuracy. The CVP and ECG waveforms are viewed simultaneously to determine the location of the *a* wave, which begins in the PR interval on the ECG. This should be distinguished from the *c* wave, which is found in the RT interval, and the *v* wave, which appears after the T wave.[37] Once the *a* wave is identified, determine the pressure at the top of the *a* wave and bottom of the *x* descent and calculate the mean to determine the CVP[36]:

$$\text{CVP estimate} = (a \text{ wave peak} + x \text{ descent base})/2 \quad (11.2)$$

The reason the mean of the *a* wave is used to estimate the CVP is because it peaks at ventricular end-diastole. Immediately after end-diastole, the tricuspid valve closes with the onset of ventricular systole, an event that follows the appearance of the QRS wave on the electrocardiogram. Therefore an alternative method of determining the CVP is to locate the R wave[37] or the end of the QRS complex[36] on the ECG. A perpendicular line is drawn at this point extending down toward the CVP waveform and where they intersect represents the true CVP.

Abnormal CVP waveform

Waveform analysis provides a method of obtaining an accurate estimate of preload in the presence of cardiovascular or respiratory disease. It can also provide important supplementary information about cardiovascular function to help diagnose or confirm the presence of certain abnormalities. Several of these situations are described in the following subsections.

Respiratory changes

Venous return varies with respiratory phase and respiratory muscle activity. During inspiration, there is a decrease in pleural pressure generated by expansion of

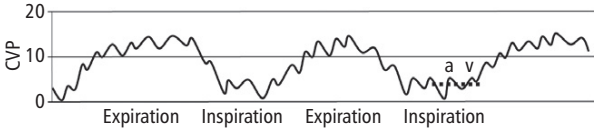

Figure 11.4 Baseline fluctuation in CVP waveform seen during forced expiration. The CVP should be estimated by calculating the mean of the *a* wave during early expiration (dashed line). The *a* wave and *v* wave are labeled.

the chest wall and caudal movement of the diaphragm.[38] This decrease in transmural pressure is transmitted across the wall of the vena cava and causes a small decrease in CVP, which returns to baseline following passive, unforced expiration. For this reason, in the spontaneously breathing, relaxed patient, the CVP reading (the mean of the *a* wave) should be measured at the end of the expiratory phase to best correspond to right ventricular end-diastolic pressure.[1,6,38]

However, when there is increased expiratory effort, which may be seen patients that are vocalizing or dyspneic, CVP will be overestimated with this method. In that situation, CVP is best obtained during early expiration prior to the onset of active abdominal muscle contraction.[6] Again, the mean of the *a* wave is used but it is necessary to evaluate the printed CVP waveform to determine the most accurate place to obtain this measurement (see Fig. 11.4). Similarly, positive-pressure ventilation may also raise the displayed mean CVP. The waveform should be examined and CVP measured from a time point corresponding to end-expiration.

Arrhythmias

Certain cardiac rhythm disturbances, such as atrial fibrillation and junctional and ventricular arrhythmias, can lead to a lack of atrial contraction and therefore loss of the *a* wave on a CVP tracing. During atrial fibrillation, there is also a prominent *c* wave resulting from overfilling of the right atrium, which is unable to generate normal contractions (see Fig. 11.5c).[37] The most accurate CVP estimate will be obtained by viewing the CVP tracing and ECG simultaneously and selecting the pressure that is present toward the end of the QRS complex, which best represents ventricular end-diastole.[26]

Ventricular premature contractions (seen as ventricular premature complexes [VPCs] on an ECG), atrial fibrillation, atrial premature contractions (seen as atrial premature complexes [APCs] on an ECG), and second- or third-degree atrioventricular (AV) node block can intermittently produce large (cannon) *a* waves due to AV dissociation, where there is a transient increase in atrial

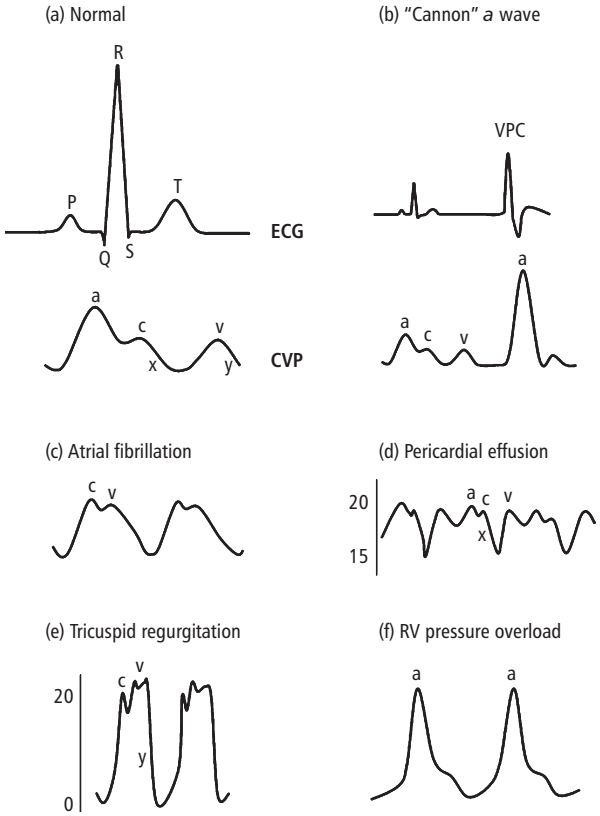

Figure 11.5 Illustration demonstrating (a) normal and (b–f) abnormal CVP waveforms. (b) Large "cannon" *a* waves are produced by simultaneous contraction of the right atrium and right ventricle and they can intermittently appear with certain arrhythmias, including second- and third-degree AV block, and some ventricular arrhythmias such as the ventricular premature complex (VPC) shown here. (c) Atrial fibrillation produces a prominent *c* wave. (d) Cardiac tamponade arising from pericardial effusion causes the CVP waveform to become flattened and there is a prominent *x* descent and small or absent *y* descent. (e) A broad and tall *c–v* wave is characteristic of tricuspid regurgitation. (f) Right ventricular (RV) pressure overload arising from pulmonary hypertension or pulmonic stenosis produces large, prominent *a* waves.

pressure caused by contraction of the atrium against a closed tricuspid valve during ventricular systole (see Fig. 11.5b).[39] When one of these arrhythmias is present, the CVP should be estimated from the normal *a* waves visible on the waveform tracing.[38]

Pericardial effusion with cardiac tamponade

Increased pericardial fluid pressure inhibits diastolic filling of the heart. This results in an increase in the mean CVP and flattening of the CVP waveform due to greater equalization of the pressures within the atria and

ventricles. A prominent *x* descent is seen due to a rapid reduction in atrial pressure during ventricular systole, and the *y* descent is small or absent (see Fig. 11.5d).[38]

Tricuspid regurgitation

Tricuspid valvular disease results in obliteration of the *x* descent during ventricular systole by a large wave created by the backward flow of blood through the incompetent valve.[38,39] This wave is composed of the merging of the *c* wave and *v* wave. Both may be clearly visible when there is mild insufficiency, but they combine to form a broad wave with a single peak when severe insufficiency is present (see Fig. 11.5e).[39]

Pulmonic stenosis and pulmonary hypertension

Conditions such as pulmonic stenosis and pulmonary hypertension can result in large *a* waves as the right atrium contracts against the elevated right ventricular pressure (see Fig. 11.5f).[39]

Alternative techniques for assessing vascular volume

The gold standard of fluid therapy decision-making would be to assess appropriateness based on its effect on cardiac output. Unfortunately, the technical challenges associated with cardiac output measurement preclude its frequent use in veterinary medicine (see Chapter 12, Cardiac Output Monitoring, for more information regarding cardiac output assessment). Due to its simpler measurement technique and clinical utility, CVP measurement has become a commonly used hemodynamic monitoring tool in veterinary critical care. However, it does not reliably correlate with cardiac output. Therefore, CVP should always be interpreted in conjunction with other clinical and biochemical markers of intravascular volume status as well as with the knowledge of the animal's cardiac and renal function.

In the critical patient, the importance of performing serial, systematic physical examinations cannot be overemphasized, as these examinations may allow the clinician to detect early changes supportive of low circulating blood volume. Compensatory cardiovascular and renal changes in response to hypovolemia result in centralization of blood volume, renal sodium and water retention, and peripheral vasoconstriction. Initially, few clinical changes will be apparent when homeostatic mechanisms are sufficient to restore tissue perfusion. However, as the volume deficit worsens, these changes typically manifest as the development of mental obtundation, pale mucous membranes, delayed capillary refill time, tachycardia, poor pulse strength, and cool extremities. When physical findings are equivocal, blood lactate concentration can provide a quantitative measure of the severity of impaired tissue perfusion and anaerobic metabolism. As shock worsens, arterial blood pressure will fall, and oliguria or anuria will develop. A high urine specific gravity will also be seen unless a concurrent disorder is present that is impairing renal concentrating ability. See Chapter 15, Monitoring Tissue Perfusion: Clinicopathologic Aids and Advanced Techniques, for more information about many of these other indices of perfusion.

In the absence of hypoalbuminemia or cardiac dysfunction, clinical signs suggestive of excessive intravascular blood volume include peripheral edema, chemosis, pleural or peritoneal effusion, serous nasal discharge, and in some cases, development of a new heart murmur or an increase in murmur intensity. Urine output is typically high and urine specific gravity low. If pulmonary edema is present, pulmonary crackles and respiratory difficulty may be evident. These findings, particularly when combined with rising CVP measurements, provide convincing evidence of volume overload.

Conclusion

In summary, the measurement of CVP is a useful adjunctive hemodynamic monitoring tool in critically ill patients and it is readily performed in any patient that has a central venous catheter. Trends, rather than isolated values, should be followed. A severely low or falling CVP suggests hypovolemia. A severely elevated or rising CVP can be seen with hypervolemia as well as with increased venous tone, reduced cardiac compliance, diminished cardiac function, or increased intrathoracic or intra-abdominal pressures. In conjunction with other clinical markers of cardiovascular function, CVP can serve as a valuable guide in the assessment and treatment of problems related to intravascular volume status and right-sided heart function.

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