How to use central venous pressure measurements

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Purpose of review

Central venous pressure is a very common clinical measurement, but it is frequently misunderstood and misused. As with all hemodynamic measurements, it is important to understand its basic principles.

Recent findings

This analysis indicates that it is best to always consider the significance of central venous pressure in the context of the corresponding cardiac output. Even more important is the use of dynamic measures to interpret the meaning of the central venous pressure. This includes the hemodynamic response to fluid load, respiratory variations in central venous pressure, and even the change in central venous pressure with changes in the patient's overall status.

Summary

The clinical application of central venous pressure measurement requires a good understanding of the concept of the interaction of the function of the heart with the function of the return of blood to the heart.

Keywords

cardiac function, respiratory variations, venous return

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Abbreviations

CVP central venous pressure
JVD jugular venous distension

1070-5295

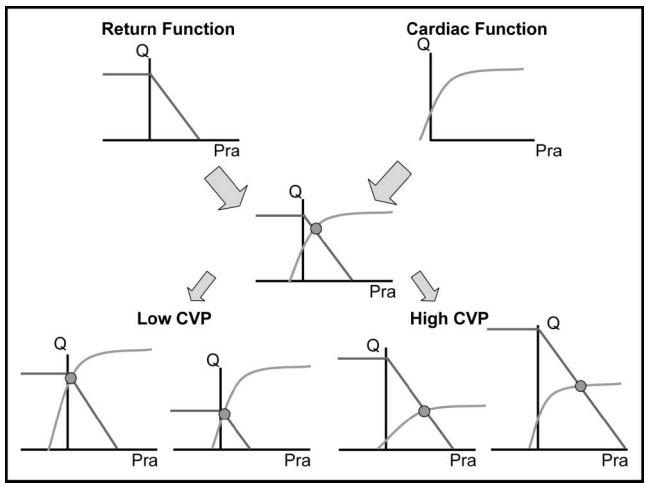
Introduction

The measurement of central venous pressure (CVP) is very common in clinical practice. The CVP can be obtained with transducers and electronic monitors, with a simple water manometer, and even by simply measuring jugular venous distension (JVD) on physical examination. In a recent survey of German intensivists, more than 90% of respondents indicated that they use CVP for the management of fluid status [1]. Despite its common use, the physiologic meaning of CVP and its clinical application are frequently misunderstood. This has resulted in many critical analyses of the value of CVP measurements [2,3]. I would argue that these are due to a lack of consideration of the true meaning of the CVP and that a proper understanding of this basic and readily available measurement can lead to very useful bedside guidelines for the treatment of patients [4–7]. In this article, the terms *jugular venous pressure*, *central venous pressure*, and *right atrial pressure* will be used synonymously because there normally is only trivial resistance along the great vessels. Some situations, however, such as sclerosis of a central vein, can increase the resistance along the major veins, and the pressures may not always be the same.

Physiologic principles

Two reasons that are commonly given for measuring the CVP are assessment of volume status and assessment of the preload of the heart, but a measurement of CVP alone cannot achieve these objectives. This is because CVP and cardiac output are determined by the interaction of two function curves: the cardiac function curve and the return curve (Fig. 1) [8–10]. The graphic analysis of this interaction demonstrates this point. CVP can be low in someone with low blood volume and normal cardiac function, but it also can be low in someone with normal blood volume and good cardiac function. For example, CVP is normally less than zero in a person at rest and in the upright posture, and even at peak exercise when the blood volume is likely high, the CVP remains in the range of 2 to 4 mm Hg [11]. Alternatively, CVP can be high in someone with decreased cardiac function and a normal blood volume as well as in someone who has normal cardiac function but who has been given excessive volume. Thus, it should be evident that a value of CVP cannot be interpreted without some idea of cardiac output at the time of the measurement. This does not have to be an actual measurement of cardiac output and could include a clinical assessment of the patent's perfusion status. In this sense, the approach to the CVP should be similar to that of the clinical assessment of the partial pressure of arterial carbon dioxide (PCO_2). When an elevated PCO_2 is observed, the next question

Figure 1. Interaction of the return function and cardiac function for the determination of right atrial pressure and cardiac output



Lower left, a low central venous pressure (CVP) can be associated with a high cardiac output and normal volume and return function or with normal cardiac function but volume and decreased return function. Lower right, a high CVP can be associated with normal return function but decreased cardiac function or normal cardiac function with high return function because of excess volume. Thus, a single value of CVP does not indicate volume status or cardiac function.

should be, what is the pH? A normal pH indicates that the patient has adapted to the increased PCO_2, and the significance is very different from that of someone in whom PCO_2 is elevated and pH is low. Similarly, CVP and cardiac output should be considered together.

Another important consideration for the proper use of CVP is the concept of physiologic limits. The cardiac function curve has a steep ascending portion and then reaches a plateau [12]. The plateau occurs because there is a limit to diastolic filling of the heart, which generally occurs first in the right ventricle. Under normal conditions, this limitation is due to constraint by the pericardium, but even in the absence of a pericardium, the cardiac cytoskeleton limits cardiac filling [13]. The limitation of cardiac filling has an important physiologic role, for it means that the heart cannot be easily overfilled and there is no downward slope to the Starling curve. Limitation of filling in the right side of the heart also limits ejection from the right side of the heart, which thereby protects the left side of the heart and the lungs. The important point in treating patients is that when the heart is functioning on the flat part of the cardiac function curve, further volume loading will not increase cardiac output. It will only increase the ventricular diastolic pressure, which has numerous negative consequences. These include an increase in ventricular wall tension and impedance of coronary flow, distortion of the ventricular septum and consequent alterations in left ventricular function, and the production of peripheral edema as well compromised flow to the liver and kidney. Thus, recognition of limitation of the right side of the heart is very important for the proper use of the CVP and will be discussed in detail below.

There is also a limitation to the return function. This occurs when the pressure inside a vessel is less than the pressure outside the vessel, which causes the soft vessel wall to collapse. The collapse does not stop the flow in the vessel, but it limits the flow, so that a further decrease in the downstream pressure, which in case of venous return is the right atrial pressure, does not result in an increase in flow. This is called a vascular waterfall because just as changes in the characteristics in the river downstream of a waterfall do not affect the flow over a waterfall, changes in pressures below the collapsed vessel do not alter flow in a vessel [14]. In a spontaneously breathing person, collapse of the great veins in the thorax occurs when the pressure inside the vessel is less than atmospheric pressure. When a person is breathing with positive pressure ventilation, however, the collapse of the veins occurs when the pressure inside the vessel is less than the surrounding positive pleural pressure, and thus collapse occurs at a CVP greater than zero [15].

Principles of measurement

Before use can be made of a pressure value, one must have an understanding of some basic principles of pressure measurement, in particular measurement in a fluid-filled system [16]. Two concepts are especially important: the concept that pressures are relative to an arbitrary reference point and the concept of transmural pressure.

When a fluid-filled catheter is used to measure pressure, as in the use of JVD, one must pick a reference point and measure the displacement of the column of blood above this point. This reference value is an arbitrary decision, but the commonly accepted point of reference for hemodynamic measurements is the midpoint of the right atrium, for this is where the heart interacts with the returning blood. During a physical examination the recommended reference point for the assessment of JVD is the sternal angle, which is where the second rib attaches to the sternum. This anatomic landmark is chosen because in the average person the midpoint of the right atrium is 5 cm vertically below this point. Importantly, this is true whether the person is supine or sitting erect up to 60°, because the right atrium is an anterior and round structure so that a vertical line perpendicular from the sternal angle points to the middle of the right atrium even when the person assumes different positions. This means that patients do not have to be supine for the measurement. This same leveling position can be used for leveling transducers for intravascular measurements, and this position is that is used in the author's unit. More commonly, pressure measurements are referenced to the fifth intercostal space on a line from the midaxilla. The advantage of this reference

point is that it does not require a leveling device to mark the position. Unlike sternal angle–based measurement, however, the midaxilla reference point should be used only when the patient is in the supine position because its relation to the midpoint of the right atrium changes with changes in body position. It is important to understand that different reference points give different values of pressure. On average, the midaxilla measurement gives a CVP value that is approximately 3 mm Hg higher than measurements based on the sternal angle [17].

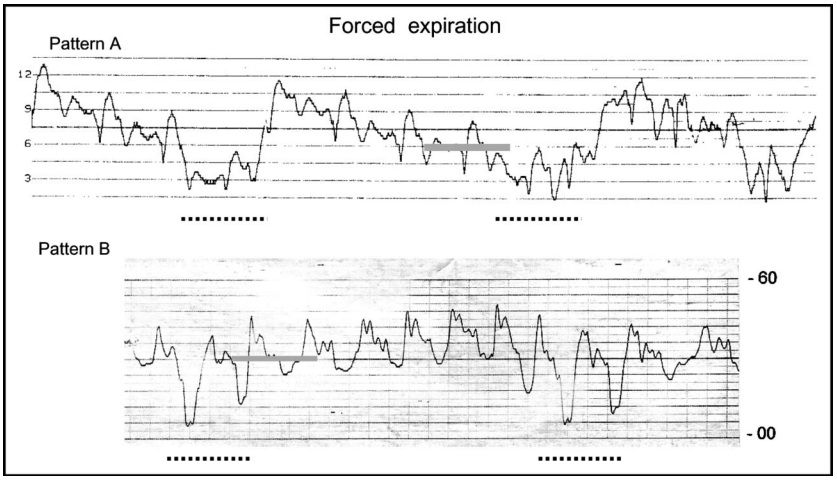
The second important concept is that of transmural pressure. The transmural pressure is the difference between the pressures inside and outside of a structure, and it is the actual pressure that distends an elastic structure such as the heart and vessels. An important problem for hemodynamic measurement is that the pressure outside the heart is pleural pressure, not the atmospheric pressure that is used to zero measuring devices. It is not possible, however, to obtain reliable measures of the pressure around the heart in patients. A good example of the effect of a change in pleural pressure on CVP is what happens to CVP during a Valsalva maneuver. CVP relative to atmosphere (including the JVD) markedly rises during a Valsalva maneuver, but the right atrium actually becomes smaller because there is a marked decrease in transmural pressure. To minimize this error, hemodynamic measurements, including the CVP, should be made at end-expiration because this is the portion of the respiratory cycle at which the pleural pressure is closest to atmospheric pressure. It is important, however, to understand that often this still does not give an adequate measure of atrial transmural pressure. Examples include patients who have either intrinsic or extrinsic positive end-expiratory pressure, mediastinal edema, pericardial fluid, or increased abdominal pressure. There is no simple solution to this problem except to recognize the potential for these conditions to lead to false conclusions about the true transmural CVP when these measurements are made.

A particularly difficult condition for the assessment of the true transmural pressure CVP occurs in patients who breathe with forced expiratory efforts. This is very frequent in critically ill patients. Active expiration increases intrathoracic pressure, as occurs with a Valsalva maneuver, and thus raises the CVP relative to atmosphere even though the transmural pressure may be decreasing. There are two general patterns (Fig. 2). In one pattern, patients forcefully expire throughout expiration but do not reach functional residual capacity (Fig. 2a). In these cases, the CVP at end expiration will be lower in respiratory cycles with longer expiratory times. The second pattern is more difficult. In these cases the person increases the force of contraction of expiratory muscles during expiration so that the CVP rises throughout expiration. When this occurs, the CVP at end-expiration is of no value, and the true CVP is likely closer to the value at the beginning of expiration (Fig. 2b).

It must be understood that although the transmural right atrial pressure is the correct value to use for the assessment of cardiac preload, it is the right atrial pressure relative to atmosphere that provides the downstream pressure for the return of blood to the heart and not the transmural right atrial pressure. This is because atmospheric pressure surrounds the veins and venules, which are the source for the return of blood to the heart. Furthermore, this means that even though some patients may respond to a fluid challenge with an increase in cardiac output at high values of CVP relative to atmosphere because of external constraining factors, the high CVP will still have important consequences for upstream vascular beds.

Figure 2. Two types of patterns of forced expiration in spontaneously breathing persons

Dotted lines indicate inspiration; solid line indicates appropriate site for the measurement. In (a), the person pushes out from the start of expiration, and the longer the expiration the lower the central venous pressure (CVP) measurement because more air is forced out. In (b), the person increases the force of expiration toward the end of expiration so that the CVP increases during expiration. The best estimate of the CVP in this patient is likely at the start of expiration, but no value is truly accurate in this situation.



Bedside examination

The CVP is essentially available in every patient through examination of the jugular veins. It is particularly worthwhile to try to make a clinical estimate of CVP before putting in central lines, for this will indicate that the electronic measurements are in the appropriate range and can sometimes give an indication of a technical problem. It will also improve one's skills in assessing jugular veins at the bedside. To compare the JVD and CVP measured electronically, it is necessary to convert the JVD to mm Hg. First, one measures the JVD above the sternal angle and adds 5 cm, which gives the pressure in centimeters H_2O. To convert this to mm Hg, one divides this value by the density of mercury, which is 13.6 and converts the centimeters to millimeters by dividing by 10. For example, a JVD of 8 cm gives a CVP of approximately 10 mm Hg. (8 + 5 = 13 cm; dividing by 1.36 gives ~10 mm Hg).

Electronic measurement of central venous pressure

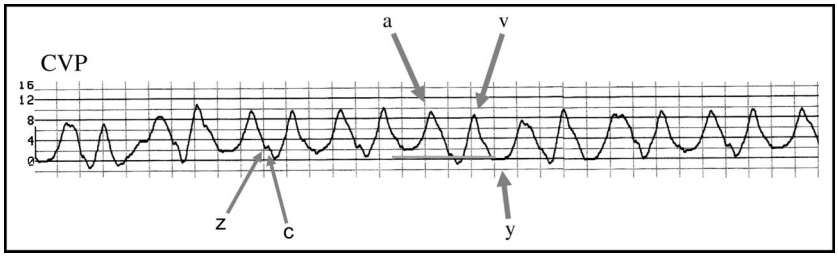
The CVP has three prominent positive waves: the 'a,' 'c,' and 'v' waves and two prominent negative waves, the 'x' and 'y' descents. The 'a' wave is due to atrial contraction, the 'c' wave is due to the backward buckling of the tricuspid valve at the onset of systole, and the 'v' wave is due to atrial filling during diastole. The 'x' descent is due to the fall in atrial pressure during relaxation of the atrial contraction. The 'y' descent is due to the sudden decrease in atrial pressure at the onset of diastole when the atrioventricular valve opens and allows the atrium to empty into the ventricle. The 'y' descent is affected by the relative filling of the atria and ventricles at the start of diastole, the compliance of the chambers, and the pressure outside the heart [18]. This last factor can be useful for marking inspiration on the hemodynamic recording in patients with spontaneous breaths, for the 'y' descent increases during inspiration. Prominent 'a' and 'v' waves raise the question where one should make the measurement on the tracing: at the top of the waves, the bottom, or the middle (Fig. 3). As in all pressure measurements, there is an arbitrariness of the measurement; however, the most common reason for assessing CVP is likely the assessment of cardiac preload. For this purpose, the best place for the measurement is the 'z' point, which is at the leading edge of the 'c' wave, for this gives the final pressure in the atrium and thus the ventricle just before ventricular contraction [19]. This value is often not easy to identify, however, in which case it can be closely approximated by the base of the 'a' wave. Timing the event from the Q wave of the electrocardiogram in turn can identify this point. It must be emphasized that this measure of CVP provides a convenient standard value that can be shared among different persons; however, there can still be important hemodynamic effects on upstream organs such as the liver and kidney from prominent 'a' and 'v' waves.

General principles of fluid responsiveness

As discussed above, it is important to be able to recognize the plateau of the cardiac functions curve because it indicates the limits of volume responsiveness of the heart. Unfortunately, the value at which this plateau occurs is highly variable among individuals. Patients can be volume limited at CVP values as low as 2 mm Hg (based on sternal angle referenced values), whereas others may respond at CVP values greater than 18 mm Hg. We recently found that 40% of patients with a CVP below 6 mm Hg did not respond to fluids [17]. Some general guidelines can be given, however. Most patients will be volume limited by a CVP of 10 to 12 mm Hg, and this range of CVP can be considered high. When giving volume challenges to patients with CVP values above this range, one should have some reason to expect that transmural right atrial pressure is less than is evident from the CVP relative to atmosphere. Possibilities include patients with high positive end-expiratory pressure, thickened right ventricular walls, high abdominal pressure, and cardiac compression by the lungs or mediastinum. When possible, however, it is best not to use a single value of CVP to predict volume responsiveness, and some kind of dynamic test should be used. It also must be emphasized that just because a patient is fluid responsive does not mean that the patient needs fluid. Normal persons will usually have an increase in cardiac output in response to a fluid

Figure 3. Measurement of central venous pressure in patient with large 'a' and 'v' waves

The preferred site is the leading edge of the 'c' wave, which is called the 'z' point. It is closely approximated by the base of the 'a' wave. Note that the difference between the top and the bottom is 8 mm Hg, so that large variations in the recorded value will occur if the position is not standardized.

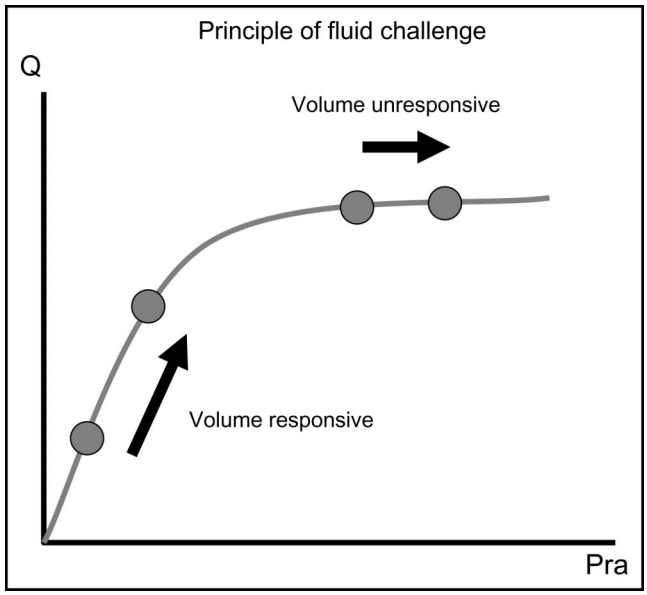


challenge, but that does not mean that they need the fluid.

Fluid challenge

The gold standard for testing volume responsiveness is to give a fluid challenge. The basic procedure is to infuse rapidly a fluid bolus of sufficient magnitude to increase the CVP by 2 mm Hg and then determine whether there is an increase in cardiac output (Fig. 4) [20]. The number of 2 mm Hg is picked because a change of this magnitude can be identified with confidence on most monitors and recording devices. The change in cardiac output should be in the range of 300 ml/min, which gives sufficient confidence of a true change with most measuring systems. To minimize the amount of fluid needed, the fluid must be given quickly. It may be necessary sometimes to use a pressure device to accomplish this aim. It is also important to remember that the change in cardiac output in response to a volume load should be immediate, for on the basis of the Starling law, an increase in end-diastolic volume increases the next stroke volume. If the fluid is given rapidly and the change in CVP is monitored continuously, the type of fluid does not matter. The faster the fluid is given, the less is needed. When a measure of cardiac output is not available, one must follow some other surrogate; however, it is worth noting that a change in arterial pressure does not correlate will with changes in cardiac output. A potentially interesting surrogate for an increase in cardiac output could be an increase in central venous oxygen saturation [21].

Figure 4. Principles of a fluid challenge



In the volume-responsive phase, a change in central venous pressure (CVP) of 2 mm Hg will produce an easily measurable change in cardiac output (Q), whereas in the plateau phase there is no change in cardiac output with a change in CVP.

Respiratory variation in right atrial pressure

In patients who have spontaneous inspiratory efforts, even if these only involve a triggering effort for a mechanical breath, the pattern of respiratory variation in the CVP can provide a very good predictor of fluid responsiveness (Fig. 5) [20]. The rationale is as follows. The decrease in pleural pressure means that the pressure environment of the heart is decreased relative to atmosphere and the return function. This is graphically represented by a leftward shift of the cardiac function curve relative to the return curve. If the return curve intersects the ascending part of the cardiac function curve, the left shift of the cardiac function curve will result in a fall in right atrial pressure and an increase in venous return and cardiac output. If the return curve intersects the plateau of the cardiac function curve, however, the left shift of the cardiac function curve will not result in a fall in right atrial pressure or a fall in cardiac output. Thus, the lack of a fall of the right atrial pressure during an inspiratory effort should mean that the heart is functioning on the flat part of the cardiac function curve and a volume load will not increase cardiac output. Of note, the test will be less predictive of an increase in cardiac output because if the heart is functioning near the plateau of the function curve, a volume infusion will have only a small effect on the cardiac output; yet, there could still be a large inspiratory fall in CVP. This concept was tested in 33 patients. Consistently with the hypothesis, volume loading increased the cardiac output in only 1 of 14 patients who did not have an inspiratory fall in CVP; in retrospect, the 1 patient who responded likely had an inadequate inspiratory effort. By contrast, volume loading increased the cardiac output in 16 of 19 patients who had an inspiratory fall in CVP [20].

'y' descent

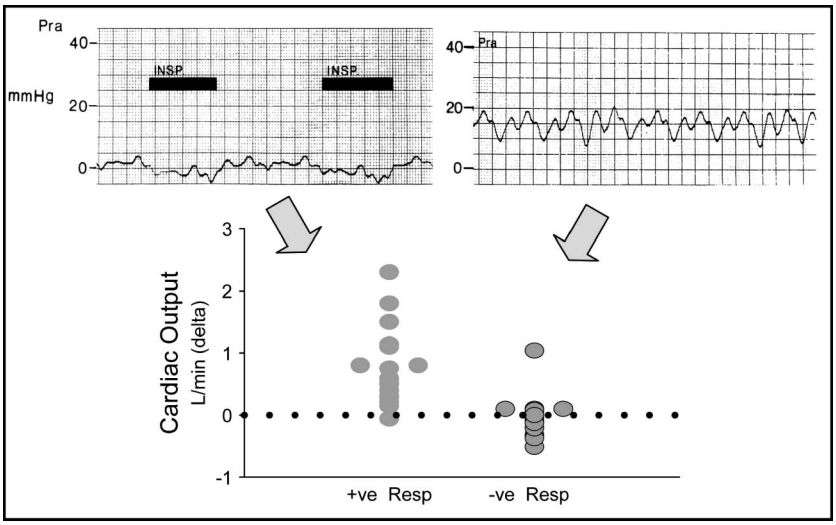
Another indicator that there will not be an increase in cardiac output with a volume infusion is the magnitude of the 'y' descent. Although the number of patients was small, we found that a 'y' descent greater than 4 mm Hg indicated that there would be no increase in cardiac output in response to fluid infusion. An explanation is that the 'y' descent is due to emptying the atrial volume during early diastole, and a steep fall means that volume must have started on the steep part of the diastolic pressure–volume curve [18].

Other patterns in the central venous pressure

The Kussmaul sign is an inspiratory rise in CVP instead of the usual inspiratory fall. The explanation for this sign is basically the same as the explanation for the use of the lack of inspiratory fall in CVP to predict a lack of response to volume loading. When the heart is functioning on the flat part of the cardiac function curve, inspiration will not decrease the CVP. The rise in pressure has two potential

Figure 5. Use of the pattern of respiratory variations in central venous pressure to predict the response to a fluid challenge

Above left, tracing from a patient with an inspiratory fall in central venous pressure (CVP). Most patients with this pattern had an increase in cardiac output. Above right, tracing from a patient with no inspiratory fall in CVP. All patients but one with this pattern had no increase in cardiac output after a volume bolus. Reproduced with permission [20].



causes. There may be a small effect from the increase in right ventricular afterload that occurs with lung inflation [22]. The more important factor, however, is likely that the increase in abdominal pressure in someone with a full venous compartment results in a transient increase in the return function, which increases CVP.

Another useful test is the hepatojugular reflux. The application of pressure to the abdomen can increase the return function and increase CVP. If the heart is functioning on the ascending portion of the cardiac function curve, the increase in preload will increase cardiac output, and the CVP will return to baseline in less than 10 seconds. If the heart is functioning on the flat part of the function curve, however, the rise in CVP will be sustained. This test thus indicates a limitation function of the right side of the heart [23].

Summary

In summary, the first step in interpreting the value of CVP is to ensure that no technical factors are spuriously affecting the measurement. These include issues related to leveling the transducer as well as factors that alter the relation of intracardiac pressures to atmospheric pressure. The issue to consider is the relation of CVP to cardiac output, or at least tissue perfusion. The most useful way to use the CVP is to observe the clinical response to a change in CVP. For example, a fall in cardiac output with a fall in CVP indicates that a decrease in return function, and volume in particular, is the likely cause of the fall in cardiac output. A fall in cardiac output with a rise in CVP indicates that a decrease in cardiac function is the likely cause of the fall in cardiac output, and treating the problem with a volume infusion is unlikely to be of great value.

Conclusion

The CVP is readily available in almost all patients. If careful considerations are given to the factors affecting CVP and its physiologic determinants, it can be of great clinical use. Detractors of this strategically placed measurement too often do not consider the physiologic context of the measurement and its potential clinical role.

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