Central venous pressure: A useful but not so simple measurement

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Objective: **To review the clinical use of central venous pressure measurements.**

Data Sources: **The Medline database, biographies of selected articles, and the author's personal database.**

Data Synthesis: **Four basic principles must be considered. Pressure measurements with fluid-filled systems are made relative to an arbitrary reference point. The pressure that is important for preload of the heart is the transmural pressure, whereas the pressure relative to atmosphere still affects other vascular beds outside the thorax. The central venous pressure is dependent upon the interaction of cardiac function and return function. There is a plateau to the cardiac function curve, and once it is reached, further volume loading will not increase cardiac output.**

Conclusions: **If careful attention is paid to proper measurement techniques, central venous pressure can be very useful clinically. However, the physiologic or pathophysiological significance of the central venous pressure should be considered only with a corresponding measurement of cardiac output or at least a surrogate measure of cardiac output. (Crit Care Med 2006; 34:2224–2227)**

Key Words: right atrial pressure; fluid administration; cardiac output; resuscitation

Central venous pressure measurements are frequently used for the assessment of cardiac preload and volume status (1). This is not surprising, considering the ready availability of central venous pressure measurements for any patient who has a central venous line. Central venous pressure can even be estimated in most people by examining the distention of jugular veins (2). However, the use of the central venous pressure is much criticized because central venous pressure poorly predicts cardiac preload and volume status (3–5). I argue that the reason for the lack of appreciation of the usefulness of the central venous pressure is the failure to consider the physiologic determinants of the central venous pressure and potential errors in measurement (6, 7).

PRINCIPLES OF MEASUREMENT

Before we assess the physiologic meaning of the central venous pressure, some basic principles of measurement need to be considered. An important point that is often not respected is that hydrostatic pressures are relative to an arbitrary reference level, and changes in the reference level change the measured pressure. The effect of leveling on the measurement of central venous pressure is particularly important because small changes in central venous pressure have large hemodynamic effects. For example, the normal gradient for venous return is in the range of 4 mm Hg to 6 mm Hg (8), and the normal cardiac function curve starts at 0 and plateaus in most people by 10 mm Hg. The commonly accepted reference level for vascular measurements is the midpoint of the right atrium, for this is where the blood returning to the heart interacts with cardiac function. As routinely taught to medical students, this can be identified on physical examination at a vertical distance 5 cm below the sternal angle, which is where the second rib meets the sternum (2). This is true whether the subject is supine or sitting up at a 60-degree angle because the right atrium is anterior in the chest and the atrium has a relatively round shape. Thus, a 5-cm vertical line from the sternal angle remains in the approximate center of the atrium even when the person is sitting upright at a 60-degree angle. This means that patients do not have to be supine for measurements when this reference level is used.

More commonly, the mid-thoracic position at the level of the fifth rib is used in intensive care units. This is easier to teach but should be used only for measurements in the supine position, because this reference position changes in relation to the mid-right atrium with changes in posture. The greater simplicity of the mid-thoracic position also likely results in less rigor in proper leveling. Values measured relative to the mid-thoracic reference level are on average 3 mm Hg greater than those based on the reference level 5 cm below the sternal angle (9).

A second important principle of measurement is that the value of central venous pressure that determines cardiac preload is the central venous pressure relative to the pressure surrounding the heart, or what is called the transmural pressure. This too is the source of a lot of measurement errors (10). The heart is surrounded by pleural pressure, and pleural pressure varies relative to atmospheric pressure during the respiratory cycle, whereas measuring devices are zeroed relative to constant atmospheric pressure. At end-expiration, pleural pressure is only slightly negative relative to atmospheric pressure, and thus the central venous pressure measured relative to atmosphere at this part of the cycle is close to the transmural pressure, whether the person is breathing spontaneously or with positive-pressure ventilation. However, in patients breathing with positive end-expiratory pressure (PEEP), transmural central venous pressure relative to atmosphere will always overestimate the transmural pressure, and there is no simple way to correct for this problem. At low levels of PEEP, however, especially in patients with decreased lung compliance, the effect is small. Furthermore, as discussed below, it is really

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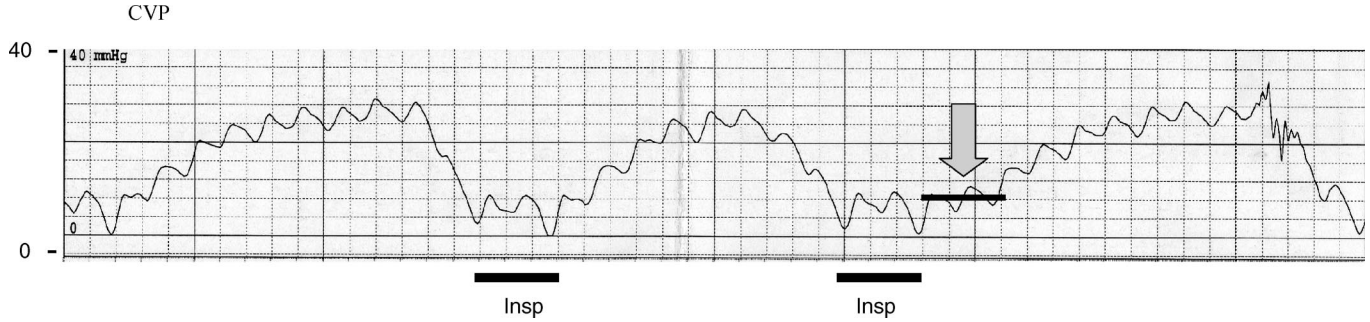

Figure 1. Example of a central venous pressure (*CVP*) tracing for a patient with forced expiration. *Insp* and the lines mark inspiration. The pressure rises throughout the expiratory phase because of transmission of pleural pressure to cardiac structures. Making the measurement an end-expiration will greatly overestimate the true central venous pressure. The digital value on the monitor will also likely be an overestimate. A reasonable guess is a measurement early in expiration, before the patient begins to push (*arrow*).

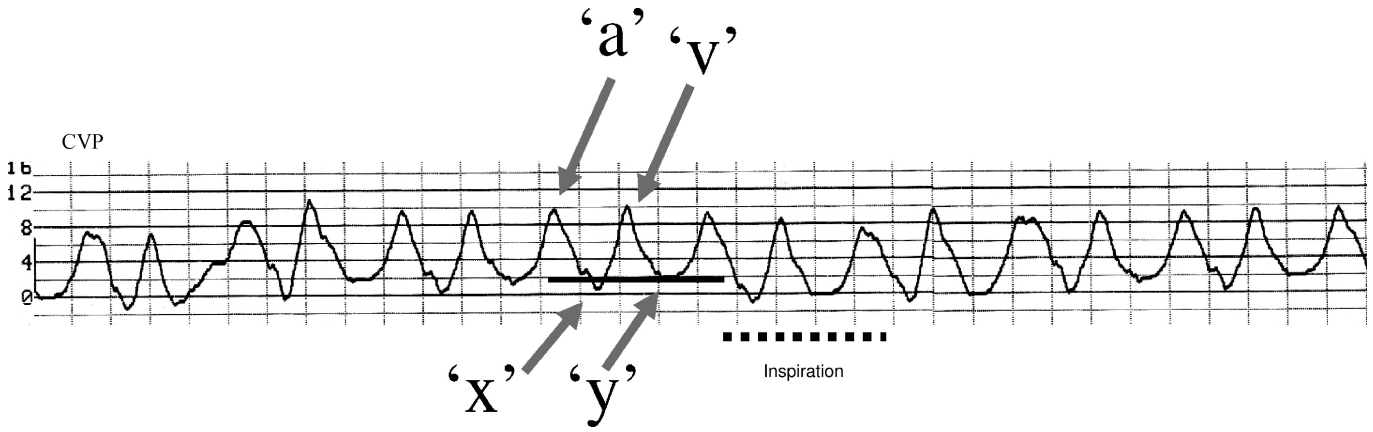

Figure 2. Example of a central venous pressure (*CVP*) tracing with prominent "*a*" and "*v*" waves. There is a small "*c*" wave after the "*a*" wave, followed by the "*x*" descent. The appropriate point for measurement is the base of the "*c*" wave (or the "*a*" wave when the "*c*" wave cannot be seen). In this example, the difference between the bottom (the correct position) and the top is 8 mm Hg.

the hemodynamic response to a change in central venous pressure that is important clinically.

Although expiration is normally passive, active expiration is very common in critically ill patients. When expiration is active, contraction of abdominal and thoracic muscles increases pleural pressure during expiration, and there may not be any phase during the respiratory cycle in which pressure measured from a transducer referenced relative to atmospheric pressure gives a close approximation of atrial transmural pressure (Fig. 1). The only thing that then can be done in this situation is to examine multiple cycles and make the measurement in a cycle where there is minimal forced expiratory effort. Sometimes, there is no value that is satisfactory, and a measurement early in the expiratory phase may be a better estimate than the value at end-expiration, but it is still a guess.

Another important consideration for the measurement of central venous pressure is where to make the measurement in relation to the normal "a," "c," and "v" waves. The "a" and "v" waves can often be in the range of 8–10 mm Hg, which means that there is a large difference in the value at the top, middle, or bottom (Fig. 2). The choice is arbitrary and each part of the cycle has physiologic significance. However, for the estimate of cardiac preload, which is the most common clinical question, the pressure at the base of the "c" wave is most appropriate because this is the last atrial pressure before ventricular contraction and therefore the best estimate of cardiac preload (11). If the "c" wave cannot be identified, the base of the "a" wave gives a good approximation. Alternatively, if the monitor has the capacity, a vertical line drawn through the Q wave of the electrocardiogram will help identify this position. On the other hand, if there is a tall "a" or "v," the peak of these waves still has hemodynamic consequences for upstream organs such as the liver and kidney. Furthermore, the central venous pressure in most dependent parts of the body in the supine position is 8–10 mm Hg higher than that measured on the basis of 5 cm below the sternal angle measurement, and this is the pressure that drives the local capillary filtration.

The central venous pressure can be estimated on physical examination by measuring the distention of the jugular veins relative to the sternal angle. One then adds 5 cm H_2O to the measured distention to obtain the central venous pressure (12). To convert the value of central venous pressure in cm H_2O to mm Hg, one needs to divide the value in cm H_2O by 13.6, which is the density of mercury compared to that of water, and multiply by 10 to convert cm to mm Hg (or simply divide by 1.36). It is worthwhile doing this before inserting central lines, for the pressure estimate will tell you that the value obtained with the transducer is in the appropriate expected range. It also improves one's skills in using the jugular venous distention to assess central venous pressure noninvasively.

DETERMINANTS OF THE CENTRAL VENOUS PRESSURE

Central venous pressure is determined by the interaction of two functions: car-

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diac function, which represents the classic Starling length-tension relationship, and a return function, which defines the return of blood from the vascular reservoir to the heart (13). Thus the central venous pressure by itself has little meaning. The central venous pressure in a normal person in the upright posture is usually less than zero (atmospheric pressure) with a normal volume and normal cardiac function (14). However, a low central venous pressure also can indicate hypovolemia or can be present in someone who is hypervolemic (i.e., with increased return function) but has a very dynamic heart. On the other hand, a high central venous pressure can be present in someone with a high volume and normal cardiac function as well as in someone with normal volume and decreased cardiac function. Thus, a central venous pressure measurement must be interpreted in the light of a measure of cardiac output or at least a surrogate of cardiac output, such as venous oxygen saturation or pulse pressure variations. The situation is similar to the analysis of Pco_2; to properly interpret the clinical meaning of Pco_2, one needs to know the pH.

USE OF THE CENTRAL VENOUS PRESSURE

Central venous pressure is commonly used to optimize cardiac preload. However, an essential point is that the cardiac function curve has a plateau and when that plateau is reached, further volume loading and increasing the central venous pressure will not alter cardiac output. The increase in central venous pressure will only contribute to peripheral edema and congestion of organs. The plateau is due to restriction by the pericardium, or in the absence of the pericardium, the cardiac cytoskeleton. A difficult problem for managing the care of patients is that the central venous pressure at which cardiac filling is limited is highly variable (3, 15, 16). It can occur at a central venous pressure as low as 2 mm Hg (measured relative to 5 cm below the sternal angle) but also as high as 18 to 20 mm Hg. However, as a working number, the cardiac function curve will plateau in most people by a central venous pressure of ~10 mm Hg (12–14 mm Hg when the mid-thoracic reference level is used) (9). When the central venous pressure is higher than 10 mm Hg and there is a question of the potential for a volume load to increase cardiac output, one should first consider possible reasons for why the central venous pressure is higher than normal. Explanations include chronic pulmonary hypertension, high positive end-expiratory pressure (whether external or internal), and some other restrictive processes.

The "gold standard" for determination of whether or not cardiac function is volume-limited is to perform a fluid challenge and determine whether an increase in central venous pressure results in an increase in cardiac output. For this purpose I recommend that there be an increase in central venous pressure of at least 2 mm Hg, for that magnitude of change can be recognized on most monitors. For the test to be positive there should be an increase in cardiac output of 300 mL/min, a value in the range of reproducibility of thermodilution cardiac output devices (17). In reality, even smaller changes in central venous pressure should increase cardiac output in someone whose heart is on the ascending part of the cardiac function curve. Consider someone in whom the plateau of the cardiac function curve occurs at a central venous pressure of 10 mm Hg and the cardiac output at the plateau is 5 L/min. The slope of the line connecting the plateau to the zero intercepts indicates that cardiac output should increase by 500 mL/min for every 1-mm Hg increase in central venous pressure, and that is still an underestimate of the steep part of the function curve. Furthermore, the increase in cardiac output should occur as soon as the central venous pressure is increased, for on the basis of Starling's law, an increase in end-diastolic volume will affect the stroke volume of the next beat.

If the clinical question is simply to determine whether the person is volume-responsive at a given central venous pressure, the type of fluid used for the fluid challenge is not important. What is important is to run the fluid in as fast as possible; the faster the fluid is given, the lesser has to be given. When I am concerned about giving too much volume unnecessarily, I sometimes use a pressure bag to increase the speed of the infusion, and as soon as the central venous pressure increases by 2 mm Hg, I measure the cardiac output.

An interesting approach to a volume challenge that can avoid extrinsic volume infusion is to elevate the patient's leg to provide an autotransfusion and observe the cardiac response (18). Another possible test is to perform a hepatojugular reflux (12). In this test the abdomen is compressed and the effect on jugular venous distention is observed. It has been shown that if jugular venous distention persists for more than 10 secs, it is indicative of right-heart dysfunction, and although this has not been directly studied, it would likely mean that the patient will not respond to volume.

The important clinical question with regard to fluid responsiveness in most patients should be phrased in the negative: "Is it unlikely that this patient will respond to fluids?" To this end, examination of the pattern of respiratory variations in the central venous pressure is useful to predict a lack of fluid responsiveness in patients who have spontaneous inspiratory efforts (15). This examination was also shown to be effective for patients who are mechanically ventilated but have at least some triggered efforts. The first step is to determine whether there is an adequate inspiratory effort. If the patient has a pulmonary artery catheter in place, respiratory fluctuations in pulmonary artery pressure give an indication of the adequacy of the inspiratory effort. If there is no pulmonary artery catheter, simple observation of the patient is often adequate. If the central venous pressure as measured at the base of the "a" wave falls by >1 mm Hg during inspiration and this is not due to the relaxation of expiratory muscles, usually the patient will respond to fluids, although some patients may not. However, the test is more important in the negative sense. If there is no inspiratory fall in the central venous pressure and a fall in pulmonary artery occlusion pressure of at least 2 mm Hg, it is very unlikely that cardiac output will increase in response to fluids.

The magnitude of the "y" descent in the central venous pressure tracing provides another potential predictor of a lack of fluid responsiveness. In a small series, we found that no patient with a "y" descent of >4 mm Hg, including the "y" descent that occurs during spontaneous inspiration, had an increase in cardiac output in response to fluids (19). However, some patients with a "y" descent <4 mm Hg also did not respond to fluids; thus, once again, a prominent "y" descent indicates that the heart is operating on the plateau of its function curve and the output will not increase in response to fluids, but a value less than this does not rule out volume limitation.

Besides the assessment of volume status, the pattern of change in central venous pressure in relation to a change in

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cardiac output can be very useful (as long as there is no major change in pleural or abdominal pressures). If a fall in cardiac output is observed, the next question to ask is what happened to the central venous pressure, because this allows an assessment of the interaction of cardiac and return functions. If cardiac output falls with a fall in central venous pressure, the primary problem is a decrease in the return function, which most often is due to a loss of stressed vascular volume; volume infusion is likely the best therapeutic approach. If the cardiac output falls with a rise in central venous pressure, the primary problem is a decrease in pump function, and therapy should be aimed at improving pump function.

Note that in all the discussion above on fluid challenges I have referred to the central venous pressure and not the pulmonary artery occlusion pressure for the management of cardiac preload. That is because the central venous pressure indicates where the heart interacts with the returning blood. Whether cardiac limitation is due to a right-heart problem or a left-heart problem, the right atrium is the place where cardiac function interacts with the return function (6). Furthermore, the right and left hearts are in series, and once the right-heart function curve reaches a plateau, changes in left-heart function will no longer affect flow, except if the change in function alters the load on the right heart and thereby alters the plateau. The expression is "no left-sided success without right-sided success." It is for this reason that I argue that the pulmonary artery occlusion pressure should never be used to optimize cardiac preload. Similarly, measurements of left ventricular size by echocardiography also should not be used to assess cardiac preload.

A very important distinction that must be made is the difference between cardiac output being volume-responsive and a patient's *need* for volume. All the discussion so far has considered how to identify volume responsiveness. The need for fluid is based on clinical parameters such as the presence of hypotension, the current use of vasopressors, and even just the need to establish volume reserves. There is a paucity of data in the literature to provide a basis for appropriate guidelines for the use of fluids for these purposes, and empirical studies are needed to provide answers.

CONCLUSIONS

The central venous pressure is there to be used by the thoughtful clinician, and as long as respect is paid to basic physiologic principles as well as principles of measurement, in my opinion it can provide a useful guide to assessment of cardiac preload, volume status, and the cause of a change in cardiac output and blood pressure.

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