Central Venous Pressure: Uses and Limitations

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Introduction

A key component of the management of the critically ill patient is the optimization of cardiovascular function, including the provision of an adequate circulating volume and the titration of cardiac preload to improve cardiac output. In spite of the appearance of several newer monitoring technologies, central venous pressure (CVP) monitoring remains in common use [1] as an index of circulatory filling and of cardiac preload. In this chapter we will discuss the uses and limitations of this monitor in the critically ill patient.

Defining Central Venous Pressure

What is the Central Venous Pressure?

Central venous pressure is the intravascular pressure in the great thoracic veins, measured relative to atmospheric pressure. It is conventionally measured at the junction of the superior vena cava and the right atrium and provides an estimate of the right atrial pressure.

The Central Venous Pressure Waveform

The normal CVP exhibits a complex waveform as illustrated in Figure 1. The waveform is described in terms of its components, three ascending 'waves' and two descents. The a-wave corresponds to atrial contraction and the x descent to atrial relaxation. The c wave, which punctuates the x descent, is caused by the closure of the tricuspid valve at the start of ventricular systole and the bulging of its leaflets back into the atrium. The v wave is due to continued venous return in the presence of a closed tricuspid valve. The y descent occurs at the end of ventricular systole when the tricuspid valve opens and blood once again flows from the atrium into the ventricle. This normal CVP waveform may be modified by a number of pathologies.

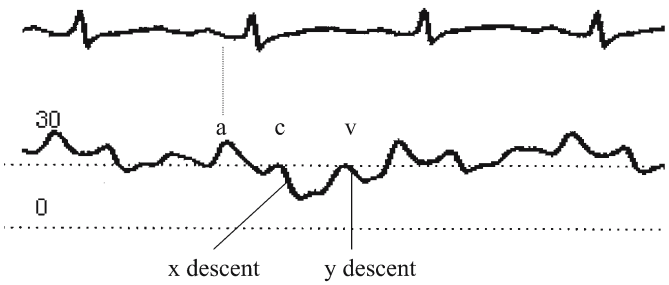

Fig. 1. Central venous pressure waveform from a ventilated patient (bottom) with time synchronized electrocardiograph trace (top). The a-wave represents atrial contraction and occurs immediately after atrial depolarization as represented by the p wave on the EKG. The c-wave represents bulging of the tricuspid valve in early ventricular systole and is followed by the v-wave, caused by atrial filling during ventricular systole.

1. In atrial fibrillation, the a wave is lost and the c wave may become more prominent; if there is coarse fibrillation of the atria, fibrillation waves may be visible in the CVP waveform.
2. In the presence of A-V dissociation or junctional rhythm where atrial contraction may occur during ventricular systole, extremely tall canon a waves occur due to atrial contraction against a closed tricuspid valve.
3. In tricuspid regurgitation, blood is ejected backwards during ventricular systole from the right ventricle into the right atrium. This produces a large fused c-v wave on the CVP trace.
4. In tricuspid stenosis, forward movement of blood from the right atrium into the ventricle occurs against a greater than normal resistance leading to an accentuated a-wave and an attenuated y-descent.
5. Similarly, if right ventricular compliance is decreased by either myocardial or pericardial disease the a-wave will be accentuated.
6. With pericardial constriction, a short steep y-descent will also be seen which allows differentiation from cardiac tamponade where the CVP will be monophasic with a single x-descent.

Determinants of Central Venous Pressure

The CVP must clearly be influenced by the volume of blood in the central venous compartment and the compliance of that compartment. Starling and co-workers demonstrated the relationships between CVP and cardiac output and between the venous return and the CVP [2, 3]. By plotting the two relationships on the same set of axes it can be seen that the 'ventricular function curve' and the 'venous return curve' intersect at only one point, demonstrating that if all other factors remain constant, i.e., if nothing happens to alter the shape of either of the two curves, a given CVP can, at equilibrium, be associated with only one possible cardiac output and, similarly, a given cardiac output (or venous return) will, at equilibrium, be

associated with a specific CVP. Both curves can of course be affected by a number of factors: total blood volume, and the distribution of that blood volume between the different vascular compartments (determined by vascular tone) will affect the venous return curve. The inotropic state of the right ventricle will affect the shape of the ventricular function curve. When any one of these factors is altered there will be an imbalance between cardiac output and venous return, which will persist for a short time until a new equilibrium is reached at a new central venous blood volume and/or an altered central venous vascular tone.

As the superior vena cava, where the CVP is measured, is a thoracic structure pressure changes in the thoracic cavity will affect the measured CVP. This has important practical implications for the measurement of CVP as the intrathoracic pressure changes cyclically with breathing. There are also important implications for the accuracy of CVP measurements in patients with either extrinsically applied or intrinsic positive end expiratory pressure (PEEP) as the intrathoracic pressure will not return to atmospheric pressure at any time during the respiratory cycle.

Additionally, as discussed in the previous section, tricuspid valve disease, myocardial and pericardial disease and cardiac rhythm abnormalities will all affect the CVP waveform.

A summary list of factors affecting the CVP is given in Table 1.

Table 1. Factors affecting the measured CVP

Central venous blood volume	• Venous return/cardiac output • Total blood volume • Regional vascular tone
Compliance of central compartment	• Vascular tone • Right ventricular compliance – Myocardial disease – Pericardial disease – Tamponade
Tricuspid valve disease	• Stenosis • Regurgitation
Cardiac rhythm	• Junctional rhythm • AF • A-V dissociation
Reference level of transducer	• Positioning of patient
Intrathoracic pressure	• Respiration • Intermittent positive pressure ventilation (IPPV) • Positive end-expiratory pressure (PEEP) • Tension pneumothorax

How is the CVP Monitored?

The CVP is commonly measured by means of a fluid filled cannula with its tip in the superior vena cava connected to either a fluid filled manometer or, more commonly in the critical care setting, to an electronic pressure transducer linked to a monitor which will display a continuous pressure wave.

In order to accurately measure CVP, it is important to appropriately set the reference level of the pressure measuring device, whether a fluid filled manometer or electrical transducer, at the level of the right atrium. In the supine patient, this point is best estimated by using the intersection of the fourth intercostal space with the midaxillary line, however, this reference may not be as accurate in patients not in the supine position [4].

If the CVP is to be used as an index of cardiac preload then, theoretically, the most relevant pressure to measure from the CVP trace is the pressure at the onset of the c wave. The c wave marks the closure of the tricuspid valve at the beginning of ventricular systole and immediately before its onset the measured pressure should be equivalent to the right ventricular end diastolic pressure (except in the case of tricuspid stenosis where a pressure gradient will always exist between the two chambers). Where no c wave is clearly visible, it is conventional to take the average pressure during the a-wave. Where no a wave is visible (e.g., in atrial fibrillation) the pressure at the Z-point (that point on the CVP wave which corresponds with the end of the QRS complex on the electrocardiogram [EKG]) should be used. It is worthy of note that many of the commercially available monitoring systems do not measure the CVP in this way but simply generate a mean CVP during the whole cardiac cycle and average this value over a number of cycles.

As can be seen from the above although CVP is used as an index of circulatory filling and preload many factors can affect the CVP waveform and the measured pressure (Table 1).

Potential Uses of the CVP

Utility of CVP to Predict Cardiac Preload

Theoretical objections

In 1895, Otto Frank demonstrated that the pressure generated in an isometrically contracting ventricle was proportional to the end diastolic volume of the chamber [5]. Starling and his co-workers expanded this work to show that the stroke volume of the contracting heart was proportional to the end diastolic volume up to a point where a plateau was reached and increasing volume would no longer increase the stroke volume (Fig. 2). It is a common practice in critical care medicine to maximize the cardiac output by using intravenous fluid administration to increase the preload and, therefore, stroke volume. However, excessive infusion of fluid carries its own problems and is therefore to be avoided; the aim therefore is

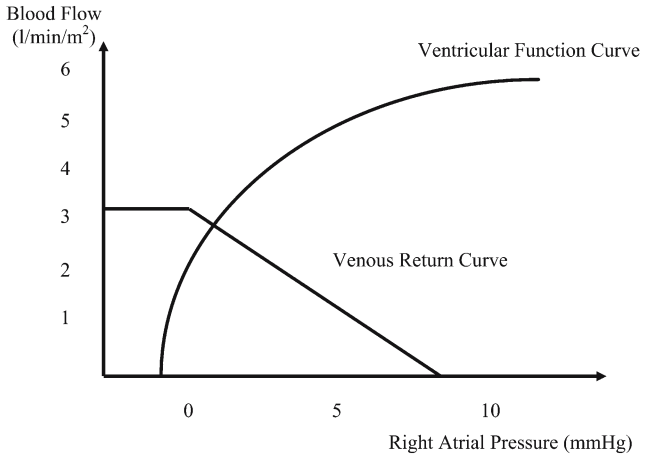

Fig. 2. Ventricular function and venous return curves

to ensure that the preload places the heart at the top of the ascending part of the Starling curve, i.e., the minimum preload to attain maximal stroke volume.

Preload is the length of the cardiac muscle fibers at the end of diastole. The use of CVP as an index of preload therefore relies on two assumptions: that CVP is equivalent to the filling pressure of the heart and that myofibril length is proportional to the cardiac filling pressure.

Unfortunately, the measured CVP often does not truly correspond to the pressure distending the right atrium at the end of diastole. As discussed above the most relevant pressure in this context is the pressure at the onset of the c wave and this is not the pressure displayed by many monitoring systems. Also, the pressure that dilates the ventricle is not the intravascular pressure but the transmural pressure, i.e., the difference between the pressure within the ventricle (intravascular pressure) and the intrathoracic pressure (extravascular pressure). Changes in intrathoracic pressure affect the intravascular pressure, for example the changes in CVP seen during the respiratory cycle, and if changes in intrathoracic pressure were completely transmitted across the vessel wall the transmural pressure would remain constant. However, it is not possible to determine for an individual patient the extent to which these pressure changes are transmitted and so the transmural pressure cannot be accurately determined. One solution would be to manually measure the end-diastolic CVP at the end of expiration and in the absence of PEEP (either intrinsic or extrinsic) when the intrathoracic pressure is equal to atmospheric pressure and the transmural pressure is, therefore, equal to the intravascular pressure. However, this is not possible with all monitors or in all patients. We would suggest that, to maximize the reliability of the measurement, where CVP is to be used to as an index of cardiac preload the end expiratory end diastolic CVP should be manually measured in the same manner that a pulmonary artery occlusion pressure (PAOP) would be measured.

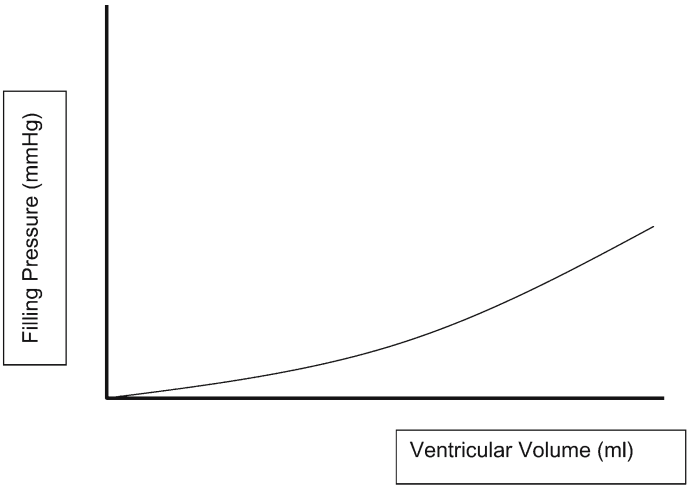

Fig. 3. Ventricular diastolic pressure volume curve

In addition, myofibril length is not linearly related to the pressure distending the ventricle. In fact the diastolic pressure/volume relationship is curvilinear, the gradient of the curve increasing as filling pressure rises (Fig. 3). This curve is not fixed between or even within individuals but will vary with factors that cause changes in ventricular compliance, e.g., inotropic state, myocardial ischemia, myocardial edema.

Clinical evidence

It is clear from the above that the CVP is not likely to provide an ideal index of right ventricular preload. This expectation is borne out in several clinical studies. CVP has been shown to correlate poorly with cardiac index [6]. Also, CVP correlates poorly with stroke volume index [7]. Given the wide intersubject variability one would expect in the ventricular function curves and venous return curves of critically ill patients, the lack of correlation of measured CVP with cardiac output in groups of patients may be considered less than surprising. However, changes in CVP during volume loading also correlate poorly with changes in stroke volume index during the same period [8-10]. In addition, the CVP correlates poorly with other indices of cardiac preload that have been demonstrated to correlate well with cardiac output or cardiac index including the intrathoracic blood volume index and left ventricular end diastolic volume index [11] and right ventricular end diastolic volume index [12, 13].

A clear problem with the use of the CVP to optimize the cardiac preload is that it does not allow optimization of left ventricular function. The left ventricular preload is related to the end diastolic left atrial pressure as the right ventricular preload is related to the end diastolic right arterial pressure. The PAOP is clinically

used as an index of left ventricular preload in much the same way as CVP is used as an index of right ventricular preload. Although in health the CVP and PAOP are closely related, the relationship between the two may be less predictable in disease [14, 15]. However, it may be argued that as the left and right ventricular outputs must be equal, the optimization of cardiac output may be adequately carried out by use of the CVP in certain circumstances as the right ventricle's output will normally stop responding to fluid infusion before the left ventricle and the left ventricle's output will clearly be limited by the right ventricle's maximum output ("no left sided success without right sided success") [16]. In clinical practice, the problem remains that a failing left ventricle may allow the rapid development of pulmonary edema after the infusion of even a small volume of intravenous fluid and this cannot be readily predicted by the use of CVP monitoring.

Utility of CVP to Predict the Volume Responsive Patient

During the optimization of cardiovascular function, an important decision is whether to attempt to increase cardiac output by giving additional fluid or whether to administer inotropic drugs. A desirable characteristic, therefore, of any index of preload is that it should be able to predict whether or not the heart is fluid responsive, i.e., whether a further increase in preload will result in an increase in stroke volume [17]. The majority of studies of the predictive value of CVP for fluid responsiveness have been unable to demonstrate a relationship between the baseline CVP and the response to filling [18-20]; those studies where a relationship between low CVP and fluid responsiveness has been demonstrated [21, 22] found such an overlap of CVP values between the responder and non-responder groups that no threshold value which would discriminate between the two groups could be determined. Use of CVP measurements to assess whether or not a patient's cardiac output will increase significantly in response to an infusion of intravenous fluid cannot therefore be recommended. Currently, the only use of CVP measurement in this process is to ensure that a large enough fluid bolus is given to attempt to increase cardiac output by ensuring that an increase in ventricular filling pressure is achieved.

Dynamic Changes in CVP

Recently there has been interest in using the dynamic changes in CVP with respiration to predict fluid responsiveness. Two studies from the same group [23, 24], both involving spontaneously breathing patients, have shown that an inspiratory fall in CVP by ≥ 1 mmHg is highly predictive of a fluid responsive cardiac index (positive predictive value 77%/84% and negative predictive value 81%/93%).

Although the CVP in the supine patient is a poor index of circulating volume postural changes in CVP may be a more reliable indicator of intravascular volume status [4]. Measurement of postural changes in CVP seems, however, unlikely to

become a widely adopted clinical tool within the context of the acutely or critically ill patient in the ICU.

Utility of CVP as a Measure of Circulatory Filling

Central venous pressure can without doubt be affected by the intravascular volume. Approximately two thirds of the intravascular volume is contained in the venous system and the total intravascular volume will affect the mean venous pressure. Only a proportion of the total intravascular volume exerts any distending force on the vasculature [25] thereby causing a positive pressure within the vasculature; this volume cannot be measured in the intact human and will vary with the vascular tone which is therefore also an important determinant of the CVP. The volume of blood in the central veins will also be affected by the distribution of the venous blood volume through the venous system: peripheral venoconstriction and the effects of the muscle pump will redistribute volume from the peripheral veins to the central veins and so increase CVP whereas peripheral vasodilatation and upright posture will redistribute volume to the peripheral venous compartment and decrease the CVP. Furthermore, the CVP depends not only on the volume of blood in the central venous system but on the compliance of that system. With so many factors other than intravascular volume affecting the CVP one might expect that CVP would be a relatively inexact measure of intravascular volume particularly in the intact organism where feedback mechanisms will compensate for a decreased intravascular volume by stimulating vasoconstriction. This expectation is borne out in clinical studies where not only has CVP been shown to correlate poorly with blood volumes measured by indicator dilution but the change in CVP after fluid resuscitation of shocked patients also correlated poorly with the measured change in blood volume [26, 27]. CVP has also been found to correlate poorly with the volume of fluid administered during ENT surgery in spite of a progressive decrease in hematocrit during surgery suggesting intravascular volume expansion [28].

Clinical Outcomes and CVP monitoring

Considering the paucity of data to support CVP as a useful physiological monitor one would not expect CVP monitoring to have a significant positive effect on outcome. There are relatively few studies that examine this issue particularly in the critically ill, presumably because CVP monitoring has become an almost routine part of ICU care.

Fluid administration targeted by CVP monitoring during hip surgery shortened the time before patients were medically fit for discharge [29]. However, similar results were obtained using Doppler flow monitoring to guide fluid administration and it might be suggested that similar results in both groups could have been achieved by simply giving larger volumes of fluid without additional monitoring. In another study, fluid administration aiming to keep the CVP greater than 5 mmHg during renal transplant surgery resulted in a greater frequency of graft

function within the first three postoperative days than in a control group without CVP monitoring [30]. Whilst these studies probably demonstrate an important use of CVP monitoring in detecting low circulating volumes in surgical patients which when detected can be appropriately managed and thus lead to improved outcome it is doubtful what bearing they have in critically ill patients where more usually the CVP is relatively high and the question is whether fluid or vasoactive drugs should be the next intervention.

In some circumstances CVP monitoring may provide prognostic information. A CVP of > 15 mmHg after cardiac surgery is a significant predictor of poor outcome [31].

Of more relevance to ICU medicine, the decrease in cardiac output in response to an increase in PEEP (from 0 to 30 mmHg) correlates with the initial level of CVP and patients with an initial CVP of ≤ 10 mmHg experience a greater fall in cardiac index than patients with CVP >10 mmHg (–30% +/– 9 vs. –8% +/– 7) [32]. Maintaining a CVP of >10 mmHg may therefore be desirable in the ventilated patient. Surprisingly the inspiratory decrease in CVP appears unable to predict the cardiovascular response to PEEP in a similar way [33].

When considering the utility of CVP monitoring it is appropriate to make the analysis in the context of other possible modalities of monitoring available to measure similar physiological variables. The most common alternative to CVP monitoring as an index of cardiac preload and volume status is pulmonary artery pressure monitoring using a pulmonary artery catheter (PAC). The use of PACs has been associated with greater morbidity and cost than the use of central venous catheters and a number of studies have suggested that in many cases they do not offer any advantages over CVP monitoring, particularly in low risk surgical patients [34] and may in fact worsen outcome increasing both the complication rate and time spent intubated after cardiac surgery [35]. An examination of the utility of PACs as an alternative to central venous catheters is outside the scope of this chapter but it is to be hoped that a clear answer to this question will be given by the large multicenter study currently underway.

Perhaps the most powerful studies indicating the usefulness of CVP monitoring, or lack thereof, in critical care are those involving goal directed therapy. One such study in septic patients showed no difference in outcome between patients with CVP or PAC monitoring where therapy was directed towards achieving normal values of measured variables; however, in those patients where therapy was directed to achieving supraphysiological values for cardiac index and oxygen delivery an improved outcome was seen [36]. Clearly such goal directed therapy requires monitoring other than simple CVP monitoring. Similarly, early goal directed therapy of septic patients in the emergency department resulted in significantly improved outcomes when therapy was directed at improving mixed venous saturations rather than at normalizing the CVP, mean arterial pressure (MAP) and urine output [37].

Conclusion

The two clinical studies on surgical patients [29, 30] confirm the potential utility of CVP monitoring in some patient groups. As a decrease in CVP is a relatively late sign of intravascular volume depletion in a patient with intact vasoconstrictor reflexes it is possible that in the patient groups in these two studies there is a significant risk of severe hypovolemia which would, if not detected by CVP monitoring, remain untreated causing increased morbidity. It may, however, be argued that CVP may be a better measure of volume status in anesthetized patients whose vasoconstrictor reflexes are pharmacologically impaired by the anesthetic drugs.

There is no convincing evidence that CVP monitoring improves outcome in the critically ill patient, particularly when other variables are being assessed. Additionally, it is clear from studies examining goal directed therapy that targeting fluid therapy to normalizing the CVP in a critically ill patient is not an optimal treatment strategy.

There is no doubt that there is a significant morbidity and possibly even mortality associated with obtaining central venous access; central cannulation having a complication rate of up to 6% even when performed by experienced staff [38]. This risk may outweigh the risk of giving large volumes of fluid without central pressure monitoring in the general surgical population. However, the majority of critically ill patients require central venous access for the administration of drugs or potassium and there appears to be some potential advantage in measuring central venous oxygen saturation at least during the early stages of treatment for which central access is also required. If central venous access is to be obtained then it would seem appropriate to monitor the CVP. As long as this variable is considered in the context of the whole clinical picture and other monitored and laboratory variables and the underlying pathophysiology taken into account then it is unlikely that CVP monitoring will lead to a worsened outcome and there are some situations such as a large occult blood loss or extreme vasodilatation where a change in CVP may provide an early warning of the problem.

The role for CVP as a monitor for use in the cardiovascular optimization of critically ill patients remains important largely because most critically ill patients will require central venous access for other reasons and so monitoring the CVP becomes essentially a risk free procedure as the risks are associated with obtaining access rather than the monitoring process itself. However, as a monitor it has significant weaknesses and with the increasing availability of other less invasive and apparently better measures of preload and circulatory filling the importance of CVP monitoring is likely to decline in this context, at least within the critical care setting, although it may be some time before other preload monitors are available on general wards in our hospitals.

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