The Use of Central Venous Pressure in Critically III Patients (Magder Book Brussels 2006)

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Introduction

The assessment of the central venous pressure (CVP) is one of the basic elements of a standard physical exam. This is done at the bedside by measuring the vertical height of the distension of the jugular veins above the sternal angle, which is where the second rib meets the sternum. The sternal angle is used because it is fortuitously approximately 5 cm above the mid-point of the right atrium whether the patient is lying down or whether the patient is sitting upright at a 60° angle. This works because the right atrium is a relatively anterior structure and sits just below the sternal angle. Differences in heart size only add a small difference to the measurement, and this standard reference point allows comparisons over time and by different operators. The midpoint of the right atrium is used as the standard reference point because it represents the lowest pressure for the blood returning to the heart, and the starting point from which the heart raises the pressure. The value is in cm H2O, which can be converted to mmHg by dividing the value in centimeters by 1.36, which accounts for the density of mercury and the conversion from cm H2O to mmHq. A major reason given for assessing the CVP is that it gives an indication of a person's volume status, but before the usefulness of this measure can be assessed, it is important to appreciate the factors that determine CVP and right atrial pressure [1]. In this chapter, I will use CVP and right atrial pressure interchangeably because there is normally only a negligible resistance between the pressure in the large veins and the right heart and the pressures are essentially the same.

What determines the CVP?

The cardiac output and right atrial pressure are determined by the interaction of the function of the heart and the function of the circuit that returns blood to the heart (Fig. 1) [2]. Cardiac function is typically represented by a 'Starling Curve', in which cardiac output is plotted as a function of the preload as estimated by the right atrial pressure. The cardiac function curve shifts upward with an increase in cardiac contractility or heart rate or a decrease in ventricular afterload and downward by the opposite. In other words, when cardiac function increases, there is an increase in cardiac output at a given right atrial pressure. The return function is determined by the stressed vascular volume (i.e., the portion of total vascular volume that creates pressure in vessels), vascular compliance, the resistance to venous return, and the right atrial pressure. The right atrial pressure is thus common to both the cardiac function and return function and right atrial pressure (or CVP) is determined by how these two functions interact. When examining the interaction of cardiac function and return function, it is important to appreciate that these two functions have limits (Fig. 1). The return function is limited when the right atrial pressure is less than atmospheric

pressure or less than pleural pressure when the pleural pressure is greater than zero. When the return function is limited, increasing cardiac function does not increase cardiac output. On a venous return curve this is seen as a plateau. The cardiac function is limited when cardiac filling is limited and therefore there cannot be an increase in cardiac output by the Starling mechanism despite increases in right atrial pressure. This is represented by the plateau of the cardiac function curve. Importantly, when cardiac function is limited, an increase in return function, as would occur with an increase in blood volume, does not increase cardiac output and can potentially do harm. The high venous pressure distends the right heart and pushes the septum to the left, which can then interfere with left heart function. High right ventricular pressures can impede coronary flow and myocardial perfusion, which can contribute to myocardial dysfunction. A high venous pressure also can compromise renal and hepatic function and lead to peripheral edema. Also of note, in the plateau phase of the cardiac function curve, decreases in right atrial pressure can occur without a decrease in cardiac output. The preload (defined as the pressure which gives the ventricular stretch before ventricular contraction) in this phase is effectively 'wasted preload'. It is thus clinically useful to know the right atrial pressure at which cardiac output becomes limited.

Unfortunately, the right atrial pressure at which the cardiac function curve reaches a plateau is very variable and exact guidelines are not possible. As an approximation, the probability of a response is low at CVPs greater than 12 mmHg [3] but, as has often been noted [4-6], the actual value of the CVP is not as useful as the dynamic response to fluids or another treatment. Importantly, it is the relationship of CVP to cardiac output that is critical. An analogous situation is the clinical interpretation of PC02 on a blood gas. To make sense of the clinical significance of a value of PC02 requires a simultaneous assessment of the pH, for only then can one determine if the process is acute or chronic and whether the person has adapted or not.



Fig.I. Right atrial pressure (and CVP) is determined by interaction of return function (upper left) and cardiac function (upper right). The abscissa is right atrial pressure (Pra) and the ordinate is flow (Q). Because the axes are the same they can be plotted together (bottom) and this gives the cardiac output and the right atrial pressure of the patient.

What does the CVP not tell you?

It is important to appreciate what the CVP does not tell you. The CVP by itself does not tell you the person's volume status or give an estimate of the mean circulatory filling pressure which is the mean pressure that distends the elastic vascular structure [2, 7]. Since the magnitude of the CVP is determined by the intersection of the pump function and circuit function, it is possible to have a very dynamic heart in association with a large blood volume and a low CVP. A simple example is exercise. During exercise, the CVP in a normal subject is only in the range of 2-4 mmHg and yet this is associated with very high cardiac outputs, and blood volume that is maximal [8, 9]. A high CVP also does not mean volume excess, for if there is a decrease in pump function or a restrictive process in the heart, the CVP could be elevated without the blood volume being increased. The volume that is important for the determination of cardiac output is the stressed volume, for this is the volume which creates the mean circulatory filling pressure, which is the driving force for the return of blood to the heart [7]. Under resting conditions, approximately 70% of blood volume is unstressed and 30% is stressed [10]. However, sympathetic stimulation can recruit unstressed volume into stressed volume and maintain a normal mean circulatory filling pressure [11]. Thus, the unstressed volume provides an internal reservoir that can maintain an adequate mean circulatory filling pressure. This means that a patient can have a reduced total blood volume and yet have a normal CVP, mean circulatory filling pressure, cardiac output, and blood pressure, because the stressed volume is maintained by contraction of capacitant veins and only the unstressed volume is decreased. However, the significance of the reduced volume and decreased capacitance (i.e., decreased total volume for the pressure) will become very apparent if sympathetic tone is decreased by sedation or if an increase in mean circulatory filling pressure is required to compensate for a physical stress such as that produced by an increase in intrathoracic pressure [12]. Furthermore, infused volume may not remain as stressed volume. An increase in cardiac output and arterial pressure could result in reflex adjustments through the baroreceptors which will result in dilation of capacitance vessels and conversion of some of the increase in stressed volume into unstressed volume [11]. The magnitude of this effect will depend upon the overall balance of the system and is not readily predictable.

Technical Issues for the Measurement of CVP

Before one can consider using the CVP diagnostically, it is essential to understand the technical factors involved in the measurement. The range of CVP at which most changes in cardiac output occur is from approximately 0 to 10 mmHg. For a normal cardiac output of 5 l/min, this means that, as an approximation, for constant cardiac function (i.e., constant heart rate, contractility, and afterload), a 1 mmHg change in right atrial pressure will result in a change of about 500 ml/min. Therefore small differences in the measurement of CVP due to differences in leveling, where the measurements are made with respect to the 'a' and 'v' waves and the assessment of the effects of respiratory efforts, can have significant impact on clinical conclusions. These issues will be discussed in this section. The most important point is that common principles are developed for this essential to allow trending of the measurements over time by the same person, repeated measurements by other health care workers, and comparison of results among different investigators.

All vascular pressures with fluid filled catheters are made relative to a reference level that is arbitrarily chosen. It is generally agreed that the mid-point of the right atrium is the appropriate standard reference point for hemodyamic measurements. This is the lowest pressure for the blood returning to the heart, and the pressure from which the heart re-circulates the blood. As noted in the introduction, the relation of the CVP to the right atrium is assessed on physical exam by making measurement of venous distension relative to the sternal angle which is a vertical distance of approximately 5 em above the mid-point of the right atrium, whether the subject is lying down or sitting upright at a 60°. This reference position is used in some intensive care units (ICUs), including my own, for leveling measuring devices, but more often the mid-axillary (mid-thoracic) position is used because it does not require a leveling device to set the transducer. It is important to appreciate that CVP measurements that use the mid-axillary position give values that are 2 mmHg greater than those that use 5 em below the sternal angle as the reference point. Furthermore, measurements made with the mid-axillary position must be made with the subject lying supine. whereas those made relative to the sternal angle can be made in any position up to 60° as long as the transducer is re-leveled.

When measuring the CVP one could choose the peak of the 'a' or 'v' wave, the mean pressure or the base of the waves' (Fig. 2). The difference between the peak and the base of these waves can be as much as 8-10 mmHg and occasionally even higher, and therefore the choice can have a significant effect on the measurement. Since a major reason for measuring the CVP is for the assessment of cardiac preload, the measurement should reflect this value. Therefore, it has been recommended that the 'z' point or base of the 'c' wave be used for the measurement for this gives the final pressure in the ventricle before the onset of systole, and therefore is the closest approximation of the preload (Fig. 2). The 'c' wave is produced by buckling of the atrio-ventricular valves back into the ventricle and the base of the 'c' wave is just at the onset of systole. However, the 'c' wave is often hard to see and the base of the 'a' wave gives a good approximation of this value. If the electrocardiogram (EKG) signal is synchronized with the hemodynamic tracings on the monitor, the onset of the onset of the sec.

QRS wave can be used to identify the appropriate point on the CVP tracing after taking into account the delay of around .08 sec of the pressure signal from the fluid filled catheter measurement compared to the electrical signal.

Fig. 2. Example of right atrial pressure tracing over two cardiac cycles. The X marks the appropriate place to make the measurement.

It needs to be appreciated that although the base of the 'a' wave is used for the measurement, this does not mean that there is no significance to the peaks or troughs of the other waves of the CVP. For example, if the base of the 'a' wave is 10 mmHg, but there is a 'v' wave with a peak of 20 mmHg, it is the value of 20 mmHg that affects the liver and kidney and can have very significant pathological consequences. Therefore, one must distinguish the measured pressure as recorded in the standard manner for reproducibility and as a measure of cardiac preload from the actual pressure that occurs during the cardiac cycle, which may have other consequences.

A major factor affecting the value of the CVP is the variation in pleural pressure for it is pleural pressure that surrounds the heart. Hemodynamic pressures are measured relative to atmosphere for this is the value that surrounds the measuring devices, and is therefore the value for zeroing measuring devices, whether the device is a column of mercury or water or a transducer. However, it is the transmural pressure, which is the difference in pressure between inside and outside the elastic structure, that determines the distension of the structure. This is not a problem when the right atrial pressure is analyzed as the backpressure for the return function, for the veins and venules that return blood to the heart are effectively surrounded by atmospheric pressure. However, the use of right atrial pressure measurements zeroed relative to atmosphere creates a problem for the measurement of pressures inside the thorax, for changes in pleural pressure relative to atmosphere effectively change the environment of intrathoracic structures relative to atmosphere and create changes in cardiac pressure which do not reflect the transmural pressure (Fig. 3). Thus, an increase in intrathoracic pressure can result in an increase is right heart pressure relative to atmosphere, even though right heart volume decreases and visa versa. This is because the distending pressure is the pressure inside the heart relative to outside the heart, but we unfortunately have to zero transducers to atmospheric pressure, which is outside the thorax, rather than pleural pressure, which is the true pressure outside the wall of the heart. To try to avoid the artifact produced by changes in pleural pressure, hemodynamic measurements are made at endexpiration, whether the subject is breathing spontaneously or whether the subject

is ventilated with positive pressure, for at end-expiration the pleural pressure is closest to atmospheric pressure.

In patients who have positive end-expiratory pressure (PEEP), there is still an added positive pressure at end-expiration that creates an error in the measurement. However, even at a PEEP of 10 cmH20, which is less than 8 mmHg, and considering



Fig. 3. The effect of changes in pleural pressure on the relationship of cardiac function and return function. The top panel shows a fall in pleural pressure as occurs with a spontaneous breath. On the left, the return function intersects the ascending part of the cardiac function curve and a fall in pleural pressure (PI) decreases right atrial pressure (Pra) relative to atmosphere and increases right heart output (flow, Q). Patients with this status should have an increase in cardiac output with volume. On the right, the return function intersects the plateau of the function curve and right atrial pressure and cardiac output do not decrease with a decrease in pleural pressure. Patients with this condition should not respond to volume. The bottom part of the figure shows a rise in pleural pressure. When the return function intersects the ascending part of the function curve (left side), an increase in pleural pressure would be expected to decrease cardiac output. When the return function intersects the plateau of the function curve (right side), an increase in pleural pressure should not decrease cardiac output until the cardiac function curve shifts enough so that the return curve intersect the ascending part of the function curve,

that less than half the PEEP is transmitted to the pleural space, and even less than that in the pathological conditions that usually require PEEP, the error is only in the range of 2-3 mmHg. Above a PEEP of I0 cmH2O, however, the pleural pressure at end-expiration can become clinically significant. An important condition where changes in pleural pressure can have a marked effect on the measurement of CVP is during forced expiration. This is a very common phenomenon in critically ill patients, whether ventilated or breathing spontaneously. The forced expiration elevates pleural pressure during expiration and thus artificially elevates the CVP. There are two patterns. In one, there is a prolonged expiratory phase; in these patients, the CVP should be measured in the longest breaths and it should be appreciated that this still may be an overestimate. In the second pattern, there is a progressive rise in CVP during expiration and in these patients, the measurement should not be made at endexpiration and a rough approximation can be made at the start of expiration before they start to bear down.

The respiratory alteration in the pulmonary artery occlusion pressure (PAOP) can give a reasonable estimate of the respiratory changes in pleural pressure however, respiratory variations in CVP do not. This is because the right atrium is connected to the large venous capacitance, which is outside the thorax. Thus the pressure source for the right atrium is not affected by the change in pleural pressure, whereas the outflow, i.e., the right atrium is directly affected. This means that the gradient for venous return is directly affected by changes in pleural pressure, which will consequently change the filling of the right atrium. In contrast, during a change in pleural pressure, the pressure in the pulmonary venous capacitance relative to atmospheric pressure changes directly with the change in left atrial pressure, so that the gradient for filling of the left heart is not affected by this mechanism. However, it is eventually affected by the serial transmission of the changes that occurred in right heart output.

How to use the CVP

The CVP measurement is most commonly used to give an assessment of the adequacy of a patient's blood volume and the preload for the right heart. However, it has been widely documented that the actual value of the CVP is a poor predictor of volume status. In reported series [4], patients with CVP of as low as 2 mmHg failed to respond to a volume infusion that increased the CVP, whereas a patient with a CVP of 16 mmHg responded (Fig. 4). In a recent series, we found that 35% of patients with a CVP of <=10 mmHg failed to respond to a fluid challenge [3]. Thus, it is not possible to give specific numbers for the best value of the CVP. On the other-hand, in the same series we found that few patients had an increase in cardiac output with volume loading when the CVP > 12 mmHg. Thus, as an approximation, volume loading is unlikely to increase cardiac output in patients with CVP > 12 mmHg unless they have something to explain the higher values such as an increase in intrathoracic pressure or a decrease in right ventricular compliance.



Initial rt atrial pressures in patients first classified as having or not having a respiratory variation in RAP with a spontaneous inspiratory effort. Although the response to fluids was very different in the two groups, the initial RAP did not predict the fluid response. The large circle marks a patient with a RAP of 2 mm Hg who did not respond to fluids and the large box marks a patient with a RAP of 16 mm Hg who responded to fluids.

Figure 4

The most specific way to determine the clinical significance of a value of CVP is to perform a dynamic test. The gold standard is to give an infusion of fluid that increases the CVP by at least 2 mmHg and then assess the change in cardiac output. The value of 2 mmHg is suggested so that one can be certain that the CVP truly increased and that there was an adequate test of Starling's law. The cardiac function curve is steep, so that a 2 mmHg rise in CVP should result in a significant rise in cardiac output (i.e., >250 ml/mins) if the heart is functioning on the steep part of the curve. If there is no rise in cardiac output with a rise of CVP of 2 mmHg, then it is highly unlikely that fluid infusion will increase cardiac output and an inotropic agent is needed to increase cardiac output. When performing a fluid challenge the rise in CVP, and not the volume given, is the important criterion for determining if the test was adequate. With the Starling mechanism, the stroke volume goes up on the contraction following an increase in end-diastolic volume. Thus, if one is concerned about the consequences of giving fluids to someone who may not have an increase in cardiac output, the amount of

test fluid can be minimized by rapidly infusing the fluid and assessing the cardiac output as soon as the CVP increases by an adequate amount. With a rapid infusion, the type of fluid should also not matter, and the faster the fluid is given the less that has to be given. It is also important to appreciate that blood pressure is not a good guide as to whether the cardiac output rose with the fluid infusion. In the series discussed above [3], we found that almost three fourths of patients who had an increase in cardiac output did not have an increase in blood pressure. It is thus very difficult to determine the rare of fluids without a measure of cardiac output or a surrogate.

Another dynamic test, involves the use of the change in CVP that occurs with an inspiratory fall in pleural pressure (Figs. 4 and 5) [4] and is based on the change in position of the cardiac function curve relative to the position of the return curve in a Guyton cardiac function-venous return plot (Fig. 3) [15]. A decrease in pleural pressure makes the pressures in the heart more negative relative to atmosphere and the rest of the body, whereas the venous return curve is unaffected because the veins are surrounded by atmosphere. When the heart functions on the ascending part of the cardiac function curve, this results in a fall in right atrial pressure relative to atmosphere and an increase in the gradient for venous return and an increase in right heart output. Under this condition, a volume infusion should increase cardiac output.

However, when the heart is functioning on the flat part of the cardiac function curve, the fall in pleural pressure does not produce a change in right atrial pressure and therefore the gradient for venous return does not change

nor does cardiac output. Under this condition a volume infusion should not increase cardiac output.

We tested this concept in 33 ICU patients (Fig. 5). A key first step was to ensure that the patients had an adequate inspiratory effort. We did this by using the respiratory variation in PAOP as an indicator of the fall in pleural pressure. A decrease of 2 mmHg with inspiration was considered to represent an adequate change in pleural pressure to test the response of the right atrium. We included ventilated patients as long as they had at least some triggered breaths, which demonstrated an adequate decrease in pleural pressure as indicated by the fall in PAOP. An inspiratory fall in right atrial pressure, measured at the base of the 'a' wave, of > 1 mmHg was considered a respiratory response. These patients were



Fig. 5. The use of respiratory variation in right atrial pressure to predict the response to a fluid challenge (adapted from [4] with permission). The top shows examples of a respiratory fall in right atrial pressure (left) or no response (right), On the bottom, as predicted most of those with a inspiratory fall in right atrial pressure had a rise in cardiac output whereas only one of those with no inspiratory fall had a rise in right atrial pressure.

expected to respond to fluids; there were 19 in this category, and 16 of the 19 had a rise in cardiac output of~ 250 ml/min with the volume infusion. It is not surprising that they did not all have an increase, because some could have started from a value of right atrial pressure that was close to the plateau. An inspiratory fall in right atrial pressure of < 1 mmHg was considered a negative response and these patients were not expected to respond to fluids; there were 14 patients in this category and of these, only 1 had an increase in cardiac output of >= 250 ml/min. In retrospect, the inspiratory effort in that patient was likely not large enough to produce a fall in right atrial pressure to properly classify the patient. The test thus is most useful in the negative. That is, patients who have no fall in right atrial pressure with an inspiratory effort, are very unlikely to have an increase in cardiac output with a volume infusion. Of importance, the initial right atrial pressure (Fig. 4), PAOP, cardiac output, and blood pressure, did not predict the response to fluids as observed in other studies [6].

We also reasoned that this test should predict which patients would have a fall in

cardiac output when PEEP is applied [16]. The reasoning was as follows. As discussed above, in patients who have a fall in CVP with a spontaneous inspiration the heart should be functioning on the ascending part of the cardiac function curve. PEEP shifts the cardiac function curve to the right of the venous return curve (Fig. 3). Thus the venous return curve will intersect the cardiac function at a higher right atrial pressure but a lower value of cardiac output, and cardiac output thus should

fall. On the other hand, in patients who have no fall in CVP with a spontaneous inspiration, the heart should be functioning on the plateau of the cardiac function curve, and these patients should have a range of increases in PEEP in which there is no decrease in cardiac output. However, when the increase in PEEP is large enough so that the ascending part of the cardiac function curve intersects the venous return curve, there will be a fall in cardiac output. We tested this in 18 ICU patients [16]. As in the study in which we tested the potential to predict the response to a fluid challenge, we separated the patients into those who had an inspiratory fall in CVP of \geq 1 mmHq and those who did not and assessed the adequacy of the inspiratory effort from the inspiratory fall in PAOP. The overall pattern was consistent with the hypothetical predictions, but there was much more variability than in the volume study. Of the seven patients who had an inspiratory fall in CVP, and therefore should have had a fall in cardiac output when PEEP was applied, the average cardiac output fell by 0.7 ± 0.8l/min (p< 0.05), but the cardiac output did not always fall. Of the 11 patients with no inspiratory fall in CVP who therefore should not have had a fall in cardiac output. the average cardiac output did not change (change of $-.04 \pm 1.5$ l/min), but the individual responses were highly variable. Thus, the overall concept was supported, but the predictions in individual patients were poor. The likely reason why this study failed to provide as strong a prediction as in the fluid challenge study, is that reflex adjustments in vascular capacitance occur when PEEP is applied, and these can maintain cardiac output in the face of increased intrathoracic pressue by shifting the venous return curve to the right [17]. The potential for this mechanism to maintain cardiac output depends on the reserves in the venous capacitance and sympathetic response, which are highly variable in critically ill patients and cannot be assessed in patients. Thus this test fails to provide an accurate predictor of the cardiac output response to PEEP but indicates the variability of the cardiac output response to PEEP. Jellinek et al. performed a similar study in which they examined the usefulness of the initial right atrial pressure, PAOP, and right ventricular volume for predicting a hemodynamic response to increases in airway pressure [18]. They found that a right atrial pressure < 10 mmHg best predicted a decrease in cardiac output. Based on our study on respiratory variation and the response to PEEP, this would indicate that they were on the ascending part of the cardiac function curve. However, although in that series 10 mmHg provided a clear cut off, I would not expect this to be true in all patients. From our prospective series [3], some patients with CVP as low as 2-4 mmHg failed to respond to fluids indicating that

they were on the flat part of the function curve and therefore should not initially have a fall in cardiac output with PEEP. Second, if the PEEP is raised sufficiently, there will be a fall in cardiac output in all patients for eventually the cardiac function curve will shift to the right sufficiently so that the ascending part of the cardiac function curve intersects the venous return curve. Finally, it should be appreciated that the transducers were referenced to the mid-thorax so that the value would have been approximately 8 mmHg if the transducers were referenced to 5 cm below the sternal angle.

A useful 'dynamic' approach to the assessment of the significance of a value of the CVP involves the use of passive leg raising to transiently increase the CVP and to then determine if this is associated with a change in cardiac output. This was used successfully by Boulain and co-workers who showed that patients who had a rise in cardiac output with leg raising also had an increase with a volume load [19]. Furthermore, an increase in the pulse pressure also correlated with an increase in stroke volume and therefore provided a potential non-invasive indication of the cardiac output response to volume loading.

In the dynamic test that used the respiratory variation in the CVP to predict the response to a fluid challenge, the patient had to have an inspiratory effort, but many patients have no spontaneous efforts. Another indicator that can be of use in these patients is the magnitude of the 'y' descent. In a small series of patients, we found that patients that had a 'y' descent of> 4 mmHg did not respond to fluids [20]. The rationale is that this represents a 'restrictive' state, which is what occurs on the plateau of the cardiac function curve. That is not to say that the patient has restrictive heart disease, but rather restrictive hemodynamics. The series was small so the specificity of this test is not well established, but it does provide a simple non-invasive guide. Another useful clinical point is that the loss of a 'y' descent in a patient with an elevated CVP, is very suggestive of cardiac tamponade.

When assessing fluid responsiveness, it is also essential to keep in mind that just because the cardiac output increases with a fluid infusion, does not mean that the person needs the fluid. In the healthy state, the heart usually functions on the ascending part of the cardiac function curve so that a volume infusion will almost always increase cardiac output, but a volume infusion is obviously not indicated. Outcome data on the role of fluids in the management of critically ill patients is lacking, and thus the decision to use fluids, even in patients who respond to volume loading, remains very subjective.

There has been discussion about whether to use the PAOP or the right atrial pressure for the assessment of the response to a fluid challenge and the preload of the heart. First of all, for both of these measures, it is not the absolute value that is important but rather the change in cardiac output that is associated with the change in their value. That having been said, I believe that the right atrial pressure is always the value that should be used when trying to optimize cardiac output by giving fluids or drugs. That is because the right atrial pressure gives the result of the interaction of the cardiac function and return function and a volume

infusion acts by changing the return function. Furthermore, the right and left hearts are in series and the only way a left sided event can affect cardiac output is by altering the right atrial pressure. Thus, even in a patient with a ruptured mitral valve or an occlusion of a circumflex coronary artery, cardiac output will only decrease when the right atrial pressure rises. Alternatively, a patient who has an underfilled left ventricle as indicated by a low PAOP, can only have an increase in cardiac output with a volume infusion if the right heart output can increase, so that if the right heart function curve has plateaued there will not be a rise in cardiac output with volume infusion. The motto is 'no left-sided success without right-sided success'. What the PAOP gives is diagnostic information. For example if the PAOP is elevated but the right atrial pressure is not, then the primary pathological process is in the left heart and diagnostic possibilities are limited to severe hypertension, mitral or aortic valve disease, or coronary artery disease, all of which can be easily assessed clinically. Equal increases in P AOP and right atrial pressure indicate a biventricular process and a right atrial pressure greater than the PAOP indicates a primary right-sided problem.

For hemodynamic management of hypotension, I begin with an assessment of the cardiac output. If the cardiac output is normal or elevated, then the hypotension is due to a low systemic vascular resistance and the primary therapy should be directed at correcting the vascular resistance. If the blood pressure fell with a fall in cardiac output, the next question is what happened to the right atrial pressure. If the right atrial pressure rose, then the primary problem is pump function and therapy should be aimed at increasing pump function. If the cardiac output fell with a fall in right atrial pressure, then the primary problem is a decrease in the return function (usually a loss of volume) and volume therapy is the likely solution.

Conclusion

In summary, CVP by itself does not indicate cardiac function or vascular volume, but rather it gives an indication of the interaction of pump function and return function. As such, a high CVP can be due to an increase in blood volume. a decrease in pump function or both, for they often go together. It is important to appreciate that the cardiac function curve has a plateau, and once the plateau is reached, further increases in vascular volume and right atrial pressure will not result in an increase in cardiac output. There is no universal value that can be given for the right atrial pressure at which the plateau of the function curve occurs, but in general the probability of the cardiac output increasing with a CVP > 12 mmHg (leveled 5 em below the sternal angle) is low, unless the patient has had a chronic increase in pulmonary pressures which altered right heart compliance or has increased pleural pressure which alters the assessment of the transmural pressure. The significance of a particular value of CVP is best assessed by a dynamic test in which the change in CVP is related to a change in cardiac output or a surrogate such as a change in pulse pressure or Doppler flows. The major clinical point is that it is not the actual CVP that counts, but the

hemodynamic consequences of a change in CVP. Reference

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