Central venous pressure monitoring

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Purpose of review

The proper use of central venous pressure requires a good understanding of basic measurement techniques and features of the waveform.

Recent findings

If attention is not paid to proper leveling of the transducer and consideration of transmural pressure then major errors are made in the use of central venous pressure. Besides the information gained from the relationship of changes in central venous pressure to changes in cardiac output, there is also much information to be obtained by examining the waveforms of the central venous pressure tracing. Examples are given of rhythm disorders, tricuspid regurgitation, cardiac tamponade, cardiac restriction, and decreased thoracic compliance.

Summary

There is much more to the measurement of central venous pressure than the simple digital value on the monitor and the actual waveform should always be examined.

Keywords

cardiac output, respiratory variation, right atrial pressure, volume management, waveforms

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Abbreviations

CVP central venous pressure
PEEP positive end-expiratory pressure

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Introduction

Central venous pressure (CVP) measurements are readily obtainable in most critically ill patients and are commonly measured [1]. I recently discussed the use of CVP measurements in this journal [2••]. In this paper I will follow up on that discussion and emphasize the monitoring aspects of CVP with more discussion on factors that affect the measurements and wave patterns. First, however, I will review some of the basic rationale for the use of CVP.

Rationale for the use of central venous pressure

CVP is determined by the interaction of cardiac function and return function and a change in either can alter the CVP [3,4]. As demonstrated by Ernest Starling [5], the CVP or right atrial pressure (for they are essentially equivalent under most circumstances) prior to the onset of systole determines the preload of the right ventricle and the ultimate output of the heart as a whole. The shape of the cardiac function curve for any preload is determined by afterload, contractility, and heart rate. The CVP also is the back pressure for the return function [3], which is determined by stressed vascular volume, venous compliance, and venous resistance. An increase in cardiac function without an increase in return function results in a rise in cardiac output with a fall in CVP [2••]. An increase in return function without a change in cardiac function results in a rise in cardiac output with a rise in CVP. The converse is true for decrease in cardiac or return functions. These simple relationships can be used to determine the nature of a change in hemodynamic status and the choice of appropriate therapy. It needs to be emphasized that generally CVP values by themselves do not provide much useful clinical information. The value must be interpreted in the context of the patient's overall status and if possible the cardiac output. For example, normal CVP in an upright person is usually below zero [6] and, by definition, the vascular volume is normal. However, high values of CVP indicate pathological processes, although – as discussed below – they do not necessarily indicate that the heart is adequately filled. Another key point for managing fluid status is that the cardiac function curve has a plateau [7] and when this plateau is reached, further increases in CVP will not increase cardiac output and constitute what I like to call wasted preload. The high cardiac filling pressures in this situation only increase capillary leak, distort the right ventricle, and potentially worsen the hemodyanmic situation [4].

It is useful to consider the magnitude of changes in CVP required for physiological effects. The force driving fluid from the periphery to the heart is the elastic recoil pressure from distended small veins and venules [3] and is called the mean circulatory filling pressure. Although are limited data from humans, based on studies that have been done [8,9] and the work of Guyton and others [10] in animals, the normal pressure gradient for venous return is estimated to be in the range of 4–6 mmHg. This means that a sudden increase in CVP of 4–6 mmHg without any reflex adjustments will reduce the return of blood to the heart to zero. On the cardiac side, the cardiac function curve plateaus in most individuals at a CVP of <10 mmHg and often even <5 mmHg (based on a reference level 5 cm below the sternal angle as discussed below). If the cardiac output at the plateau is 5 l/min and a simple tangent is drawn from 0 to 10 mmHg, the slope is 500 ml/min per mmHg, which is an underestimate of the steep part of the cardiac function curve. In this case, a 1 mmHg increase in CVP should produce an increase of >500 ml/min cardiac output. From this discussion, it should be evident that there needs to be great care in making the measurement of CVP. Factors affecting the measurement will be discussed in the next section.

Factors affecting the measurement of central venous pressure

Factors that affect hydrostatic measurements include zeroing, leveling, where on the waveform to make the measurement, and actual pressure across an elastic structure (transmural pressure).

Zeroing

The first and foremost concept that needs to be understood is the relative nature of hydrostatic pressure measurements [11]. To begin, pressure transducers are opened to air so that an assessment of the deviations from atmospheric pressure can be made. Atmospheric pressure is thus the zero value. This means that a CVP of 10 mmHg at an atmospheric pressure of 760 mmHg is actually 770 mmHg. If the absolute pressure were used rather than the value relative to atmosphere, changes in atmospheric pressure of only 10 mmHg would produce seemingly large hemodynamic changes and one would have to continuously check with the weather office to determine if atmospheric pressure or the patient's pressure has changed!

Leveling

Pressure measurements made with a fluid-filled system are also relative to an arbitrary reference level (Fig. 1). On physical examination, the standard reference level for assessment of jugular venous distension and pressure is the sternal angle, which in most people is ~5 cm vertically above the mid-point of the right atrium. This is true even when the person sits up at a 60° angle because the right atrium is an anterior and relatively round structure so that the mid-point remains at the same vertical distance below the sternal angle. In our unit we use this reference level for hemodynamic measurements (Fig. 2). To do this we place a carpenter's level on the sternal angle and level the transducer 5 cm below the level of the sternal angle. When the patient sits up the transducer is re-leveled to the new position. The proper level to use on the transducer is the level of the stopcock because this is where the transducer is opened to the atmosphere for zeroing and takes into account the fluid column that it necessary to fill the transducer. The difference between the stopcock and bottom of a standard transducer can be almost 4 cm, which would result in a pressure variation of ~3 mmHg between a pressure referenced to the top compared with a pressure referenced to the bottom. More commonly, the mid-thoracic position at the fourth interspace is used to level the transducer. This level gives

Figure 1 Illustration of the principle of leveling and relative pressure

The level of the measuring device is at the bottom of the tank in A and the pressure is 20 cmH_2O. The level is at the top of the tank in B and the pressure is 20 mmHg whether the channel from the bottom or top is open. Atmospheric pressure pushes down on all the surfaces so that water will be at the same level in the tank and the manometers.

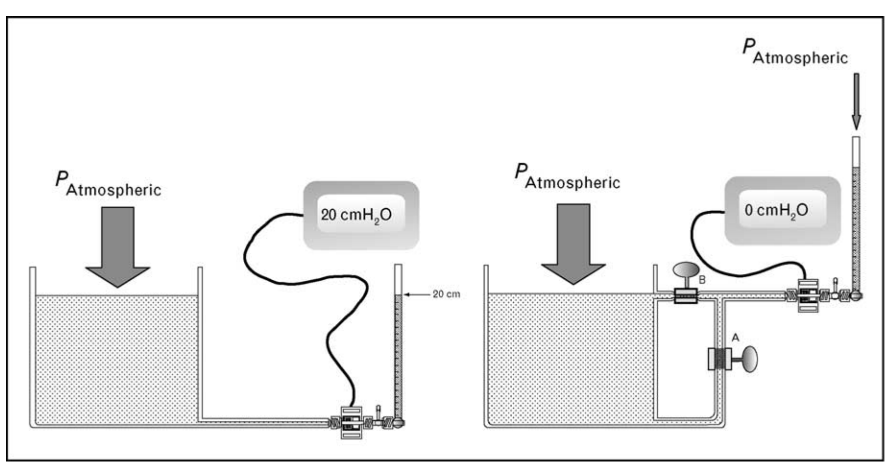
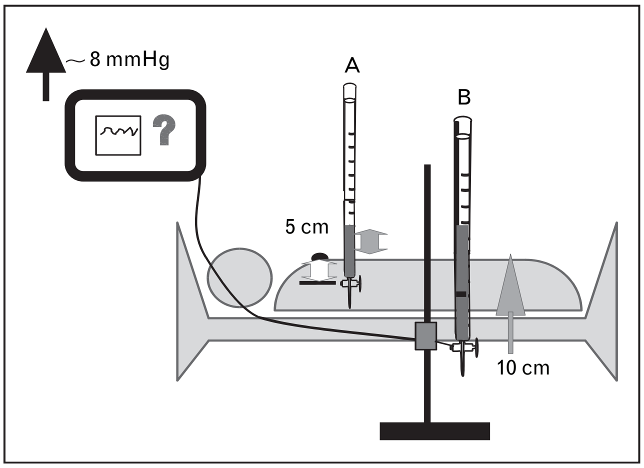


Figure 2 Illustration of the effect of lowering the bed by 10 cm on the measurement of central venous pressure with a fixed transducer position



Proper leveling of the transducer for measurement of the central venous pressure is 5 cmH_2O below the sternal angle. This is represented by the base of manometer A. When the transducer is fixed to the wall or a pole, raising the bed is like lowering the base of the manometer (B) relative to the mid-point of the right atrium and is 'seen' as an increase in measured pressure of approximately 8 mmHg (10 cm/1.36).

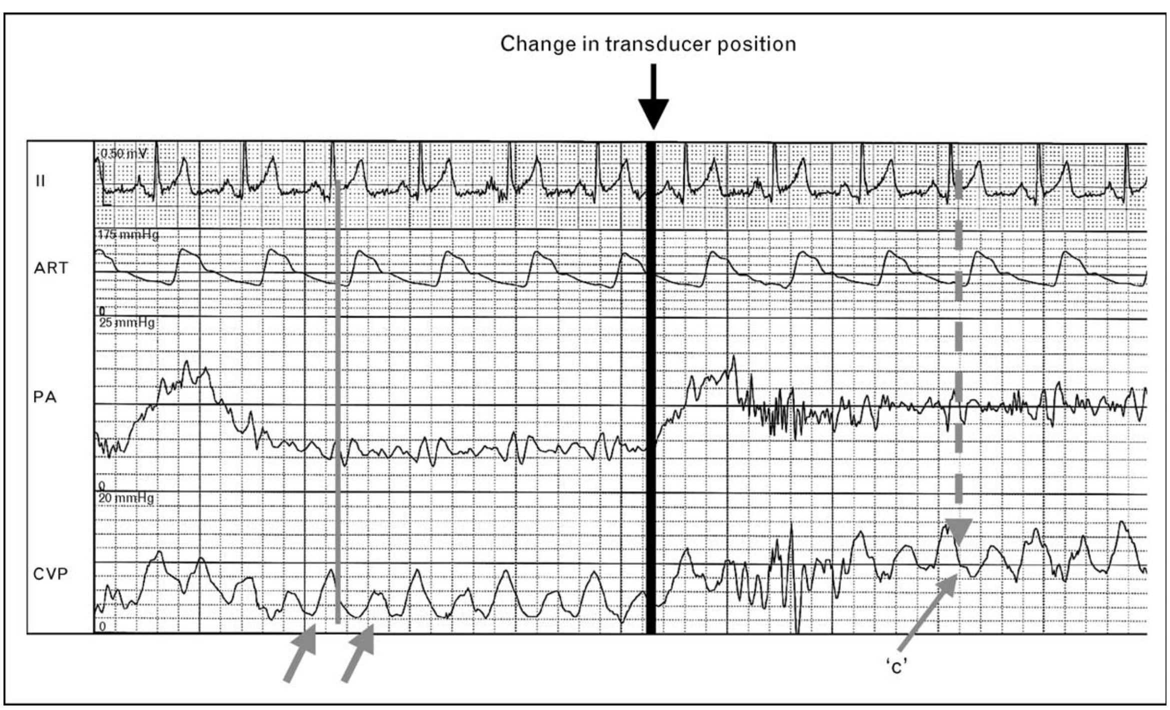
values that are on average 3 mmHg greater than pressures measured relative to 5 cm below the sternal angle. This needs to be considered when comparing results from different investigators. Furthermore, the mid-axillary position should only be used with patients in the supine position because the relationship of this level to the mid-right atrium varies in different postures.

Figure 3 shows an example of lowering the transducer by 10 cm relative to the bed. This would be the equivalent of keeping the transducer in the same position and raising the bed. It appears that there was a sudden large change in CVP but there was simply a change in bed position. The transducer now 'sees' an extra column of 10 cm of fluid which results in an ~8 mmHg change in the CVP (1 mmHg=1.36 cmH_2O; Fig. 2). At the initial level of 2 mmHg one might have given fluids whereas at the value of 10 mmHg one might have considered a diuretic. An important clue that this was due to a change in the level of the transducer relative to the patient is that the change in pulmonary artery wedge pressure was exactly the same as the change in CVP because they were on the same manifold. Note however, that the change is not as obvious in the arterial tracing because the 'gain' of the tracing does not make the change evident. When there is a large change in CVP the level of the measuring device should always be checked.

Where to make the measurement

The tracing in Fig. 3 also brings up the problem of where to make the measurement on the waveform. If the

Figure 3 Example of lowering the transducer by 10 cm



The top line shows the rhythm strip, the second is arterial pressure, the third is pulmonary artery occlusion pressure (P_{pao}), and the fourth central venous pressure. The a, c, and v waves on the central venous pressure are marked. Note that a is greater than v. The thinner vertical lines mark the c at beginning of systole and the appropriate place to measure central venous pressure.

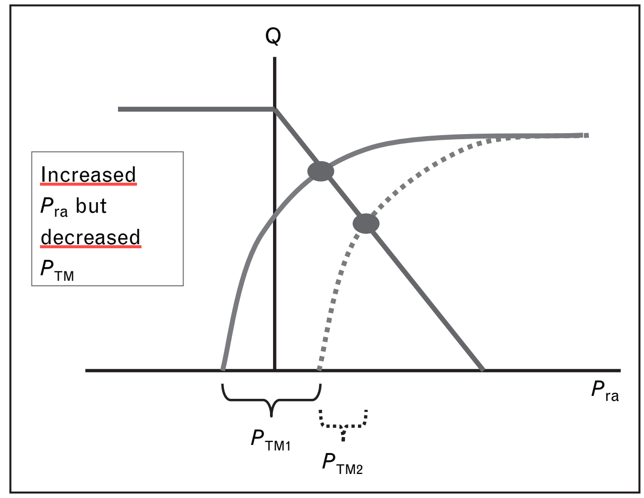
bottom of the tracing is used at end expiration the value is 2 mmHg, if the top is used it is 9 mmHg, and the in the middle it is 5 mmHg. To answer the question of where to make the measurement one first needs to identify the waves. In this example the most prominent positive deflection is the a wave and the v wave is the smaller positive deflection. The x and y descents are of similar magnitude. A primary purpose for measuring CVP is the assessment of cardiac preload. This is best measured at the base of the c wave which is not readily evident in most of the beats of this tracing but can be seen in the part of the tracing after the transducer was moved. The c wave represents the buckling of the atrio-ventricular valve into the atrium during the start of systole. The base of the c wave is used because this is the final pressure in the ventricle before the onset of contraction and represents the final distending force for the ventricle, which is preload. When the c wave is not evident, the base of the a wave usually gives a similar estimate, but in this example it is not clear either. The solution then is to use the QRS wave to time the events and note the CVP value associated with the QRS of the electrocardiogram signal (adjustments may have to be made for the delay in the fluid signal compared to the electrical signal). In this example, the arterial pressure tracing is a good indicator of the timing of events. I would have said that the CVP is 4 mmHg based on a line drawn from the onset of the arterial pulse and the evident c wave a beat after the transducer was moved. The ideal solution for such cases is to obtain a paper record so that users can compare their arbitrary decisions. Changes from this agreed-upon measurement are then what is most important.

Transmural pressure

Another key factor to keep in mind is that the pressure that is important for the distention of an elastic structure such as the heart and blood vessels is the transmural pressure of the structure. This is defined as the pressure difference between inside and outside the structure. The measurement problem is that although the heart is surrounded by pleural pressure, which differs from atmospheric pressure during the respiratory cycle, we are restricted to making pressure measurements relative to atmospheric pressure. To minimize this problem, pressures are made at end expiration when pleural pressure is closest to atmospheric pressure, but there are important limitations that need to be considered. First an increase in positive end-expiratory pressure raises pleural pressure at end expiration, which means that the measured value always deviates from the true transmural value (Fig. 4).

However, it is worthwhile considering some quantitative aspects which put the clinical significance of this in perspective. In someone with normal lungs about half the airway pressure is transmitted to the pleural space and less is transmitted when the lungs are diseased. Thus a

Figure 4 Illustration of the difference between right atrial pressure (P_{ra}) relative to atmosphere and transmural pressure (P_{TM})

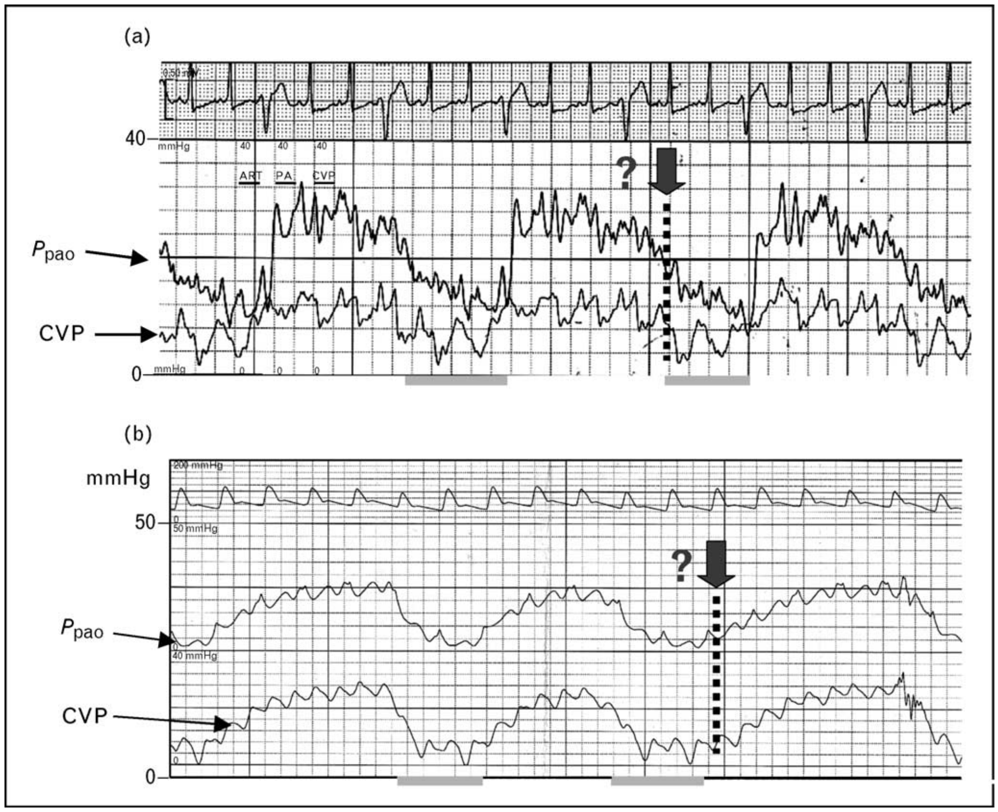


The figure shows the cardiac function/venous return curves. An increase in pleural pressure shifts the cardiac function curve to the right relative to atmospheric pressure. There is a rise in P_{ra} relative to atmosphere but a fall in P_{TM} and a fall in cardiac output (Q).

positive end-expiratory pressure (PEEP) of 10 cmH_2O, whether extrinsic or instrinsic, would likely increase the pleural pressure by about 4–5 cmH_2O and even less in someone with stiff, diseased lungs. Thus, a PEEP of 10 cmH_2O, likely increases the measured CVP by less than 3 mmHg in most persons. However, the effect of PEEP can become significant at higher values of PEEP. It should, however, be remembered that the hemodynamic response to a change in CVP does not require knowledge of the absolute value and the cardiac output response to a change in CVP is still of use. On the other hand, it is the CVP relative to atmosphere and not the transmural right atrial pressure that determines the back pressure for venous return and the force determining capillary filtration in extrathoracic vessels.

The distinction of transmural pressure versus pressure relative to atmosphere becomes very important in patients who breathe with a forced expiration. Forced expirations can have two general patterns. In one the patient forcibly expires from the start of expiration and the pleural pressure progressively decreases during the expiration (Fig. 5A). In these cases the CVP value at the end of a long breath gives a good approximation of the true CVP. In the second pattern the patient increases the force of expiration throughout the expiratory phase so that the pressure continuously rises during expiration (Fig. 5B). In these patients the end-expiratory value of CVP gives a completely erroneous estimate of the transmural CVP. This pattern is very common in critically ill patients and must be watched for. Because of this problem, I would recommend never making a CVP

Figure 5 Two examples of forced expiration in spontaneously breathing subjects



In A the top line of the tracing shows the rhythm strip, and the bottom part the overlap of pulmonary artery occlusion pressure (P_{pao}) and central venous pressure (CVP). There is a decrease in P_{pao} and central venous pressure throughout expiration. Inspiration is marked by the thick lines. In B, the P_{pao} and central venous pressure are shown without overlap. There is a progressive increase throughout expiration. The dotted lines and arrows mark the suggested place of measurement but in reality there is no accurate place. Note that in B end expiration would give a completely false estimate of transmuaral pressure (P_{TM}) because of the sustained increase in thoracic pressure, as evident on P_{pao}.

measurement without first observing the pattern of the waveform on the monitor and assessing the inspiratory and expiratory components.

Information from the central venous pressure waveform

Besides the actual pressure measurement there is much information that can be gained from examining the waveforms.

The a and v waves

Typically the a wave is greater than or equal to the v wave in the CVP tracing of a normal person [12]. Figure 6 shows a tracing from a young woman who had a major bleed following a hysterectomy. The a wave and x decent are prominent with smaller v wave and y descent. Also note that the y and x descents get deeper with a spontaneous inspiration; this can be used to time inspiratory events [13]. With positive-pressure ventilation they get smaller. In patients following cardiac surgery the a wave is often smaller than the v wave and is likely due to decreased right ventricular function [12].

Tricuspid regurgitation

The atrial waves give an indication of competence of the tricuspid valve. A particularly striking example of tricuspid regurgitation is shown in Fig. 7. This woman had a post-partum cardiomyopathy and had just undergone a mitral valve repair. There is a small a wave and a massive v wave that begins with the onset of systole, which is consistent with the v wave being due to tricuspid regurgitation rather than filling of a noncompliant atrium. This CVP pattern would make insertion of a pulmonary artery catheter very difficult because it would be hard to recognize when the catheter has crossed the tricuspid valve. Note that the pulmonary artery tracing is much narrower and starts from a higher pressure.

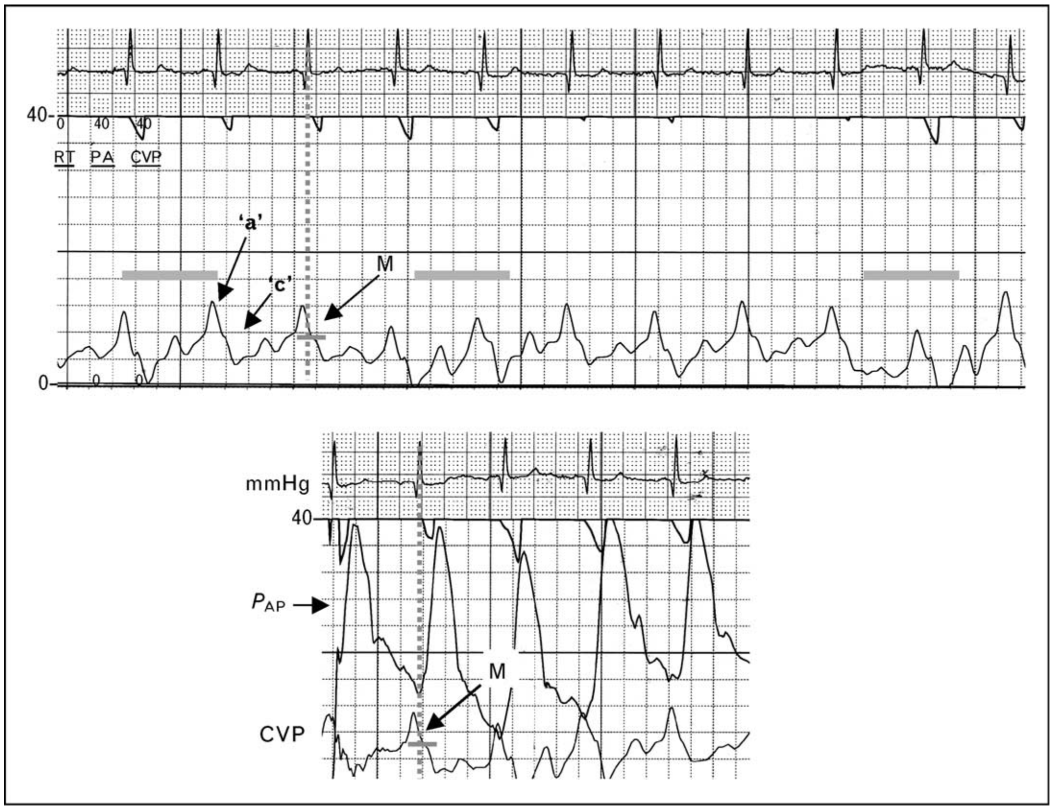
Rhythm disorders

Recognition of the wave pattern can be helpful for interpreting rhythm disorders. Figure 8 shows the CVP tracing of a patient with episodes of ventricular tachycardia. This patient had undergone a mitral valve repair and coronary artery bypass. He was breathing spontaneously and atrially paced. He had prominent v waves with ventricular premature contractions and a burst of v waves with four beats of ventricular tachycardia.

Intermittent cannon a waves can indicate the presence of pacemaker syndrome. In Figure 9 the patient is ventricular paced but the patient's own sinus beats march through the cycle because the sinus node is not inhibited

Figure 6 Patient with large a wave

M marks the appropriate place for measurement. The bottom tracing shows the overlap tracing with pulmonary artery pressure (P_{AP}). This also can be used for timing the end of diastole and the place to make the measurement. CVP, central venous pressure.



by the ventricular contractions. When the atrial contractions coincide with right ventricular systole and a closed tricuspid valve a large a wave is produced that interferes with normal right-heart filling and lowers the cardiac output. This patient's cardiac index improved when the pacemaker was turned off.

Figure 7 Patient with large 'v' waves in the central venous pressure (CVP) tracing

This indicates severe tricuspid regurgitation. The line marks the appropriate place to make the measurement. However, it must be appreciated that the peak of the v wave, which is 35 mmHg, will still have an important impact on upstream structures such as the liver and kidney. P_{AP}, pulmonary artery pressure.

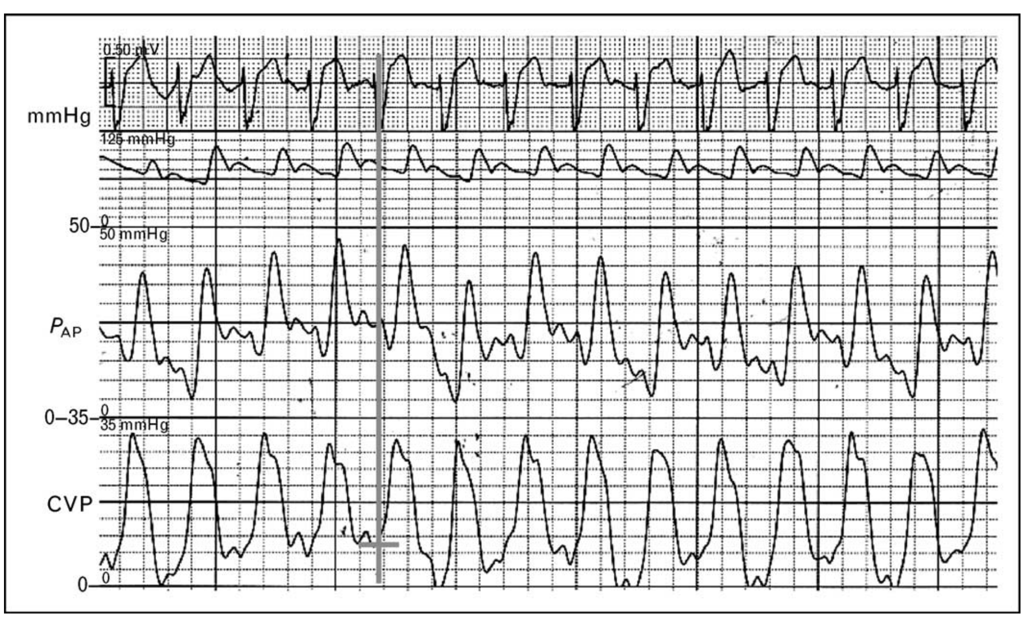


Figure 8 Example of tall v waves on the central venous pressure (CVP) tracing of a patient with short runs of ventricular tachycardia and frequent PVCs

The circles and arrows mark ventricular beats.

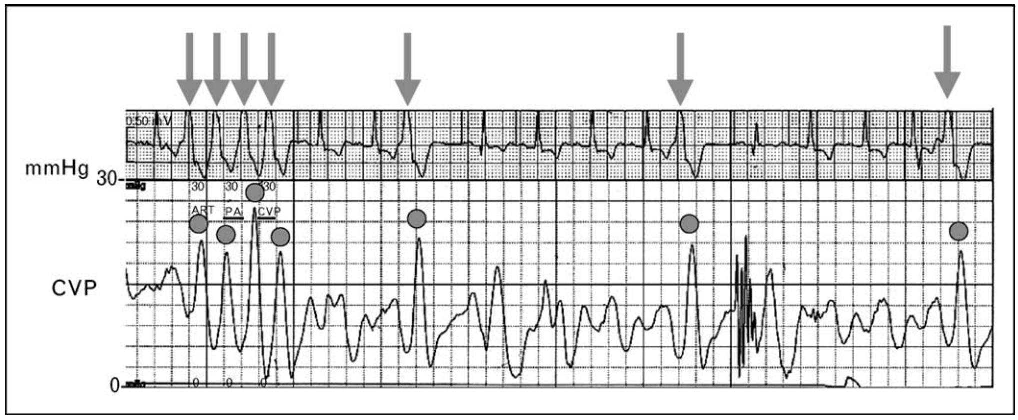


Figure 9 Example of pacemaker syndrome in a patient with ventricular pacing and atrio-ventricular dissociation

The top tracing shows the central venous pressure (CVP) with intermittent cannon a waves and the bottom tracing shows the central venous pressure with the rhythm strip. Circles mark the p waves and arrows mark the onset of a waves. Note that as the p waves march into the QRS produced by the pacemaker, tall a waves appear and then diminish as the a marches further. ECG, electroencephalogram.

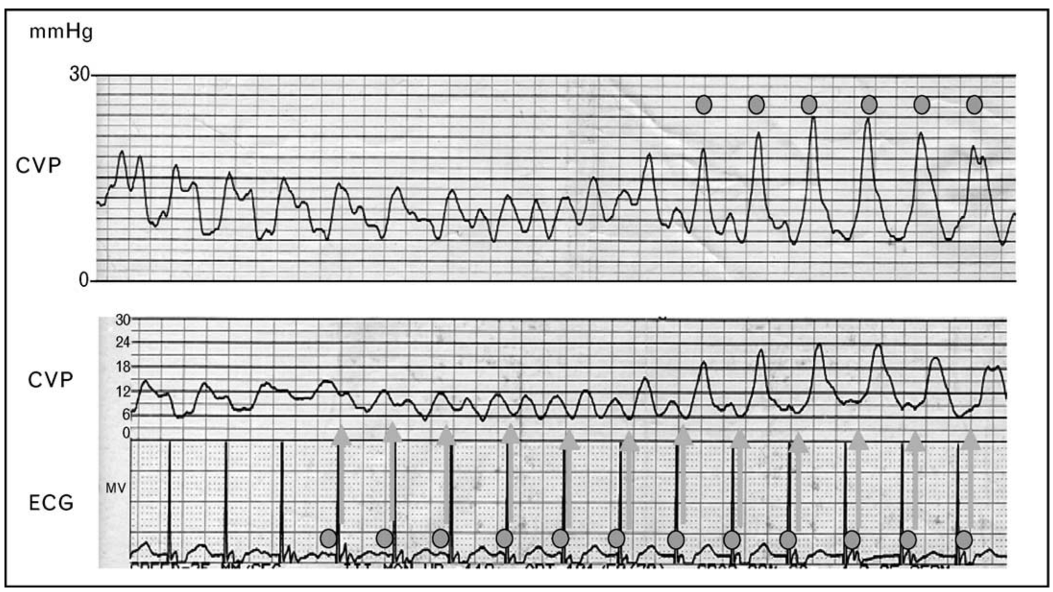


Figure 10 Example of a large y descent which indicates that this patient is likely volume-limited

There is also a rise in the central venous pressure (CVP) at the end of the first expiration which is likely a forced expiration. Note that the y descents become larger on inspiration, which can be used to mark inspiration.

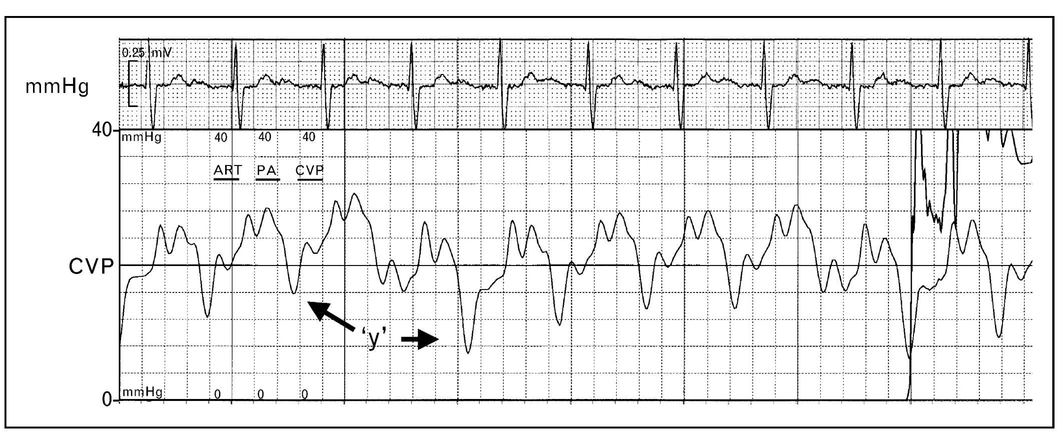
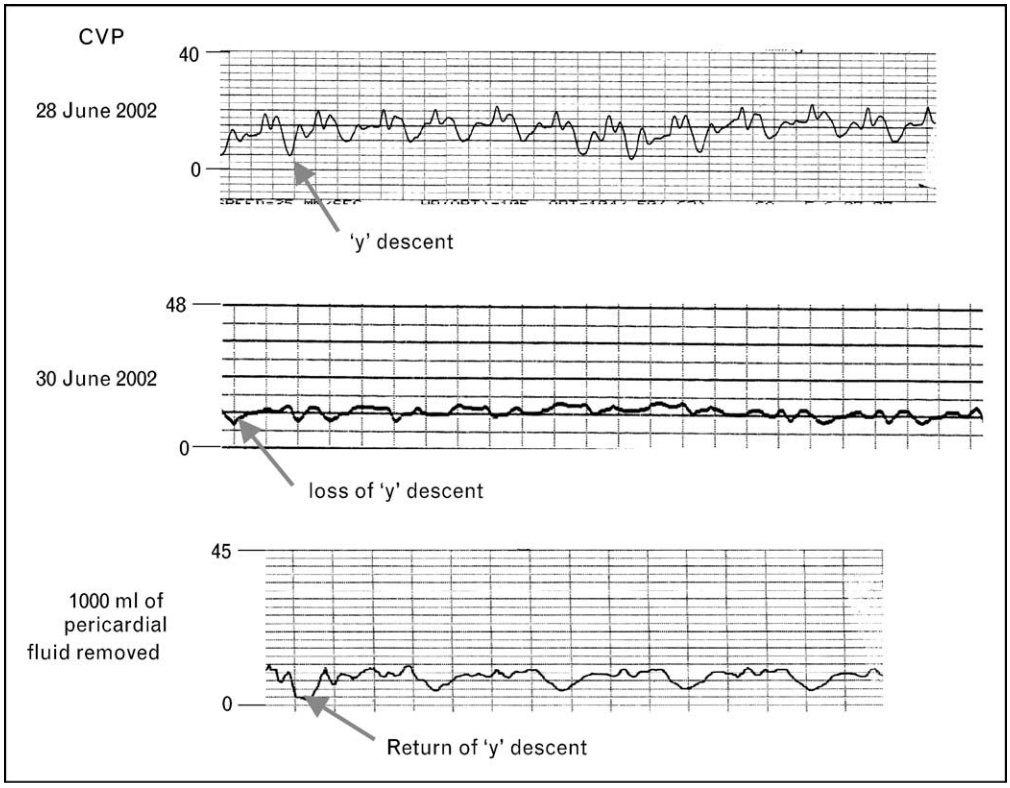


Figure 11 Example of loss of y descent in a patient with cardiac tamponade following cardiac surgery

In the top tracing (28 June 2002) a y descent is present. On 30 June his pressure drops and his norepinephrine requirement increases. There is a loss of the y, indicating tamponade, which was confirmed by echocardiography. In the lower tracing, 1000 of pericardial fluid was removed. His pressure rose and the y descent returned. CVP, central venous pressure.



The y descent

The presence of a large y descent indicates restriction of right ventricular filling (Fig. 10) [13]. This can be due to intrinsic stiffness of the ventricular wall or occur in a ventricle that is excessively volume-loaded. In either case, further volume loading is unlikely to change cardiac output. We found that when the y descent is >4 mmHg the patient is unlikely to have a rise in cardiac output in response to a volume challenge [13]. Whereas this general observation is likely valid, the sample size was not large so the precise cutoff value has yet to be determined.

Tamponade

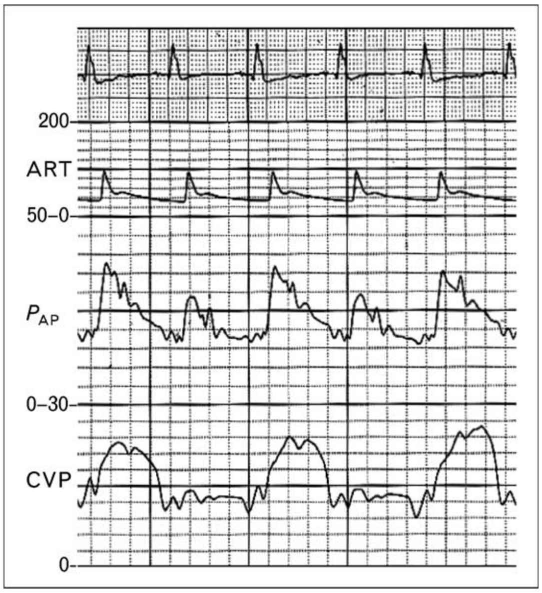
Equalization of right and left atrial pressures suggests either cardiac tamponade or a restrictive process. These diagnoses can be distinguished by examining the x and y descents. Loss of the x and y descent argues strongly for tamponade, whereas the presence of a prominent y descent argues against tamponade (Fig. 11). The reason why the x and y descents are lost in tamponade is that the pericardial fluid keeps the pressure inside the pericardium constant. The only time that fluid can enter the pericardial sac is during sytole when the ventricles empty and allow the atria to fill. During diastole this volume is transferred to the ventricle, again maintaining constant pericardial volume and pressure. There is thus no period where pericardial pressure falls and thus no x or y descent.

Respiratory variation in central venous pressure

We previously showed that the respiratory pattern in CVP can be used to predict fluid responsiveness [14]. We argued that patients who have no inspiratory fall in CVP are on the flat part of their cardiac function curve and will not respond to fluids whereas patients who have an inspiratory fall in CVP are on the ascending part of the cardiac function curve and may or may not respond to fluids, depending how close they start to the plateau. Patients with an inspiratory fall in CVP are also more likely to have a fall in cardiac output with the application of PEEP [15].

Clinically useful information can also be obtained from the magnitude of the respiratory swings in CVP. In spontaneously breathing individuals a large fall inspiratory fall in CVP indicates that there was a large fall in pleural pressure. Unfortunately the lack of a fall does not indicate that there was not a large inspiratory fall in pleural pressure and is the basis of the test of volume responsiveness discussed above. The reason is that if the heart is operating on the flat part of the cardiac function curve then there is essentially no compliance to the walls of the heart so that changes in pleural pressure are not

Figure 12 Example of a patient with decreased thoracic compliance due to marked abdominal distention and ischemic bowel following liver resection



The top of the tracing is the rhythm strip, the second line is arterial pressure (ART), the third line is pulmonary artery pressure (P_{AP}) and the fourth line is central venous pressure (CVP). Note the marked inspiratory rise in central venous pressure and P_{AP} with mechanically induced inspiration.

transmitted to the heart. Another way to think of this is that a trivial increase in the volume of the heart can maintain the pressure relative to the atmosphere. A fundamental difference between the effects of inspiration on the right and left hearts is that the right heart is connected to a reservoir that is surrounded by atmosphere so that a fall in pleural pressure increases the return of blood to the right heart. In contrast the left heart is fed by a reservoir that is in the thorax so the environment of the left heart does not change relative to its reservoir.

In patients who are ventilated with positive-pressure ventilation the magnitude of the rise in CVP can give an indication of thoracic compliance (Fig. 12). Unless the patient is ventilated with very high inspiratory pressures, a large inspiratory rise in the CVP suggests that the compliance of the thoracic wall (which includes both the chest and abdomen) is decreased. In that case the inspiratory rise in CVP is due mainly to the pleural pressure increase and the large pleural pressure increase indicates that the inspiratory force is not being dissipated through the chest wall. This can be an important clue to the development of an abdominal compartment syndrome.

Conclusion

The CVP cannot be taken as a simple measurement. It must be considered in the context of the patient's overall hemodynamic status and how that status changes with changes in the CVP. Small changes can be significant so that great care must be taken in making the measurements. By always observing the waveforms and not just looking at the digital values, errors in measurement can be avoided. There is also a lot of useful clinical information to be obtained from just examining the waveforms.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:
- • of special interest
- •• of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 000–000).

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