Arterial and Central Venous Pressure Monitoring

James A. L. Pittman, MD, BSc, FRCA

John Sum Ping, MD, FRCA

Jonathan B. Mark, MD

"… the information that the pulse affords is of so great importance and so often consulted, surely it must be to our advantage to appreciate fully all it tells us, and draw from it every detail that it is capable of imparting." F. A. Mahomed, 1872[1]

The pulsatile arterial pressure wave is a fundamental clinically monitored variable that is used commonly to determine heart rate and rhythm and to provide the principle cardiovascular parameter indicative of adequate tissue perfusion. Detailed analysis of the pressure wave reveals that it has more to offer than this, and careful observation of its more subtle characteristics can provide additional useful diagnostic information. It is therefore surprising that it is given so little attention. There is a similar tendency to overlook the added information obtainable from close analysis of the central venous pressure (CVP) waveform. In both measurements, clinicians tend to concentrate on the numeric values rather than the more qualitative data that are available. The shape and timing of the pressure waves and the changes that occur during the respiratory cycle should not be overlooked.

In the first half of this chapter, we describe how some additional clinically important information can be obtained easily from close study of the arterial pressure waveform. The second part of the chapter has a similar focus on the CVP wave. We begin by identifying some technical considerations relevant to all pressure monitoring that are necessary to ensure that the high-resolution monitoring displays now available in the operating room and intensive-care unit can be readily and correctly used for this purpose. A clear understanding of the properties and limitations of monitoring systems is necessary to ensure accurate analysis of pressure waveforms.

Reprints: Jonathan B. Mark, MD, Anesthesiology Service (112C), Veterans Affairs Medical Center, 508 Fulton Street, Durham, NC 27705 (e-mail: mark0003@mc.duke.edu).

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■ Technical Considerations For Direct Pressure Measurement

Arterial cannulation with continuous pressure waveform display remains the accepted reference standard for blood pressure monitoring of hemodynamically unstable patients. The arterial pressure waveform is a complex wave that represents the summation of a series of mechanical pressure signals of different frequencies. It has a characteristic periodicity, termed the fundamental frequency, which equals the pulse rate.[2] The waveform displayed on the bedside monitor can be derived from Fourier analysis and described as the summation of a series of waves that are multiples or harmonics of the fundamental frequency. In clinical monitoring systems, 6 to 10 harmonics are required to provide a clinically acceptable reproduction of the true intraarterial pressure waveform (Fig. 1).[3]

Monitoring systems are designed to have dynamic response characteristics that allow accurate reproduction of pressure waveforms across the wide range of heart rates and frequency contents of arterial pressure waves commonly recorded in clinical practice. The monitoring system will have its own natural resonant frequency, and it is important that this does not overlap the harmonic frequencies present in the monitored pressure waveform. When this occurs, the monitoring system will resonate, and the output pressure signal will be distorted and exaggerated.

Although similar technical issues apply to the measurement of CVP waveforms, these are not typically a problem in clinical practice. Unlike arterial pressure waveforms, CVP waves do not contain high-frequency components. As a result, the pressure measurement system does not require a high-frequency response to measure the CVP waveform accurately.

The dynamic response characteristics of catheter transducer systems not only depend on system frequency response, but also the system-
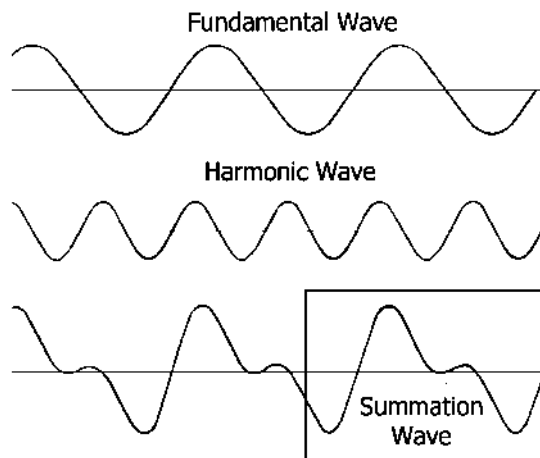
Figure 1. *The arterial pressure waveform as a sum of sine waves by Fourier analysis. Summation of the top and middle sine waves produces a waveform with the morphologic characteristics of an arterial blood pressure trace.*

damping coefficient that describes the absorption of oscillatory energy by frictional forces. A monitoring system must be adequately damped so that it dissipates energy produced by the components of the measurement system, reducing potential artifacts that could distort waveform morphology. If a system is overdamped, signal definition will be lost, and the pressure peaks and troughs will be attenuated, although mean arterial pressure values remain reasonably accurate. In clinical practice, most monitoring systems are underdamped but have a natural frequency that is sufficiently high that the effect on the monitored waveform is limited.[4]

The pressure monitoring system should consist of a short length of stiff tubing free of air and clot, thereby assuring that the system will have an acceptable natural frequency that exceeds 12 Hz. Problems arise when air bubbles or blood clots become trapped in the connections or tubing of the monitoring system. These expose the patient to risk of embolism and distort the measured pressure signal in an unpredictable fashion. A small air bubble can lower the natural resonant frequency and cause the monitoring system to resonate or ring, resulting in a spuriously elevated systolic blood pressure. On the other hand, a large air bubble will lead to excessive signal damping and cause underestimation of the true systolic blood pressure.[5]

The natural frequency and damping coefficient of a measurement system can be evaluated to predict when pressure signal distortion is likely. This is done conventionally using the "fast flush test," which determines the dynamic response of the measuring system through an examination of the artifact that follows a pressurized flush of the manometer system.[6,7] The natural frequency and damping coefficient of the system can then be determined (Fig. 2).[8] A plot of natural frequency and damping coefficient provides a visual display of the dynamic response characteristics of catheter-tubing-transducer monitoring systems and a means to determine if the system has the physical characteristics that will result in accurate pressure waveform measurement. (Fig. 3).[4] Note that the higher the natural frequency, the wider the range of damping coefficients that will still provide an accurate arterial pressure waveform. In clinical practice, it appears that most arterial pressure monitoring systems are underdamped, and this leads to the systolic arterial pressure "overshoot" typically seen in arterial pressure traces.[9]

In summary, interpretation of invasive pressure measurements requires consideration of the dynamic response of the system, and an understanding and assessment of damping and natural frequency. If the dynamic response of the system is suboptimal, the pressure measurement will be affected, leading to an incorrect diagnosis of hypotension or hypertension. Limiting pressure tubing length and stopcocks, avoidance of air bubbles, clots, or catheter kinking will improve the signal and the accuracy of pressure measurement. Because most clinical pressure monitoring systems are underdamped, direct intraarterial systolic pressure val-
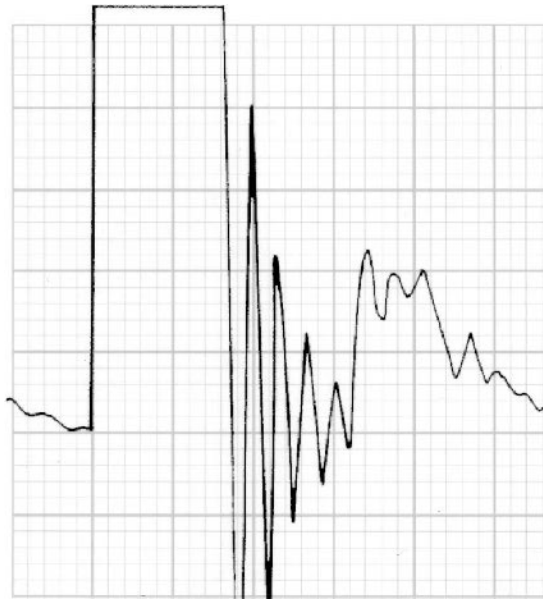
Figure 2. *The arterial pressure artifact resulting from a fast flush test allows calculation of the natural resonant frequency and the damping coefficient of the monitoring system. The time between adjacent pressure peaks determines the natural frequency and the height or amplitude ratio of adjacent peaks allows determination of the damping coefficient.*

ues generally exceed those obtained by noninvasive measurement methods. Mean arterial pressures measured by direct and indirect methods should remain relatively comparable and are often more appropriate for clinical decision-making.

Pressure Transducer Setup

Before invasive arterial or venous pressure monitoring, the pressure transducer must be zeroed, calibrated, and placed in the appropriate position relative to the patient. Intravascular pressures are referenced against ambient atmospheric pressure by exposing the pressure transducer to air through an open stopcock and pressing the zero pressure button on the monitor. Transducer calibration is not required with the current generation of disposable transducers that are designed to meet acceptable standards for accuracy. On the other hand, positioning of the pressure transducer is crucial and, in the authors' experience, represents the part of the transducer setup process most prone to error. The transducer must be leveled or horizontally aligned with a specific position on the patient's body that represents the upper fluid level in the chamber or vessel from which pressure is to be measured. It is important to recognize that when fluid-filled catheter systems are used for pressure measurement, this horizontal level is the only factor that contributes to the measured pressure. In contrast, the position of the catheter tip within the chamber or vessel does not influence the pressure reading.[10]
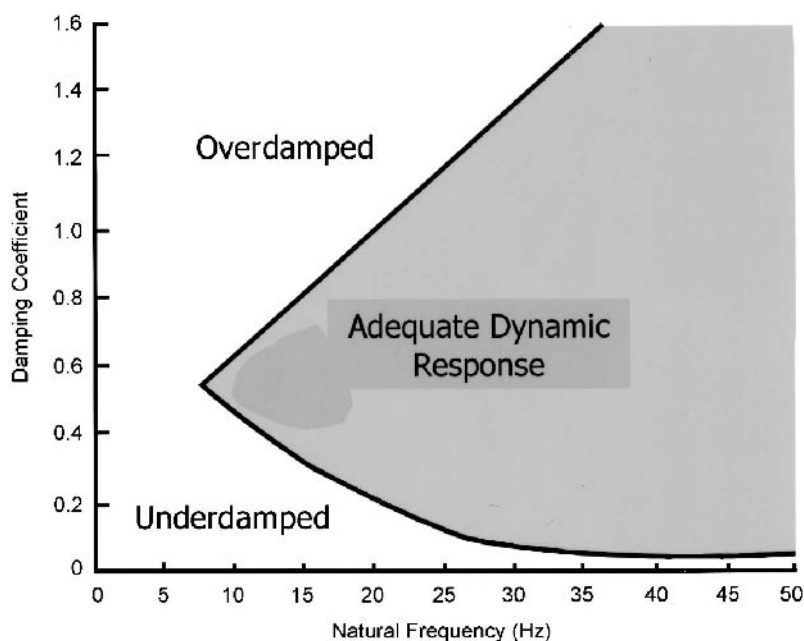
Figure 3. *The relation between the natural frequency and damping coefficient. Monitoring systems that have dynamic response characteristics that fall within the shaded area will provide accurate pressure waveforms.*

Proper positioning of the pressure transducer is critical for measurement of CVP and other central vascular pressures because small errors in transducer height will have a proportionally large effect on the measured cardiac filling pressure. In view of these considerations, CVP transducers should be positioned 5 cm posterior to the left sternal border at the fourth intercostal space, because this point better represents the upper fluid level of the right atrium compared with the more commonly used transducer alignment at the midaxillary line. In the typical patient, transducer alignment at the midaxillary line will result in a CVP overestimation of approximately 5 mm Hg.

Although these same considerations are relevant to the measurement of arterial blood pressure, the precise location of the arterial pressure transducer is less important, owing to the relatively large pressures being measured relative to the magnitude of the potential error in pressure measurement from an improperly positioned transducer. When a patient is being monitored in the sitting position, however, transducer height becomes a significant consideration. If the transducer is placed at the standard thoracic level, the arterial pressure at the level of the heart will be recorded. However, monitoring of cerebral perfusion pressure requires that the transducer be placed at the level of the brain approximat-

ing the position of the Circle of Willis. As expected, the arterial pressure at the level of the brain will be considerably lower than the pressure at the level of the heart in the sitting patient.[11]

Indications for Direct Arterial Pressure Monitoring

Standard indications for direct arterial pressure monitoring fall into 5 general categories.

1. Failure of indirect measurement
2. Arterial blood sampling
3. Intentional pharmacologic or mechanical cardiovascular manipulation
4. Continuous real-time monitoring
5. Supplementary diagnostic clues

Careful analysis of the arterial pressure waveform can give important information regarding the condition of a patient. High-resolution displays now available in the operating room and intensive-care unit can readily be used for this purpose.

Complications of Direct Arterial Pressure Monitoring

Considering the fact that arterial cannulation is an invasive vascular procedure, it is surprisingly safe and well tolerated. The incidence of distal ischemia is probably in the range of 0.1% or less,[12] and it is unlikely that the size or composition of the arterial catheter is a major determinant of complication rate. Complications of arterial catheterization are less common than with central venous catheterization, and arguably, the most frequent complication of arterial catheterization is equipment misuse and misinterpretation.[13] This serves to highlight the importance of correct operator knowledge, training, and experience in the safe application of invasive monitoring modalities. In general, manual or automated noninvasive blood pressure monitoring should be used in addition to direct arterial pressure monitoring to confirm the results and identify situations when there are monitoring problems.

■ Arterial Pressure Waveform Morphology

The shape of the arterial pressure waveform is generated by the ejection of blood from the left ventricle followed by the subsequent peripheral arterial runoff (Fig. 4). The waveform starts with a steep systolic upstroke during systolic ejection that should follow the electrocardiographic (ECG) R-wave by approximately 120 to 180 ms. The waveform reaches a peak and
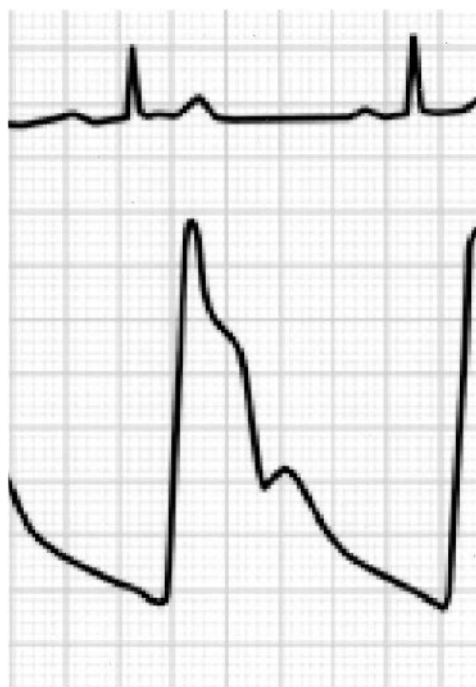
Figure 4. *Normal arterial blood pressure waveform recorded from the radial artery and its relation to the electrocardiogram.*

then starts to decline as left ventricular ejection decreases. End systole is marked by closure of the aortic valve, which inscribes the dicrotic notch or incisura in the central aortic pressure trace. When the arterial pressure wave is measured peripherally from the radial artery, as performed typically in clinical practice, the dicrotic notch has a smoother appearance, appears later in the cardiac cycle, and its timing only approximates aortic valve closure. Instead, this feature of the peripheral arterial waveform results more from pressure waves reflected from the periphery of the arterial vascular bed. The arterial pressure wave continues to decline following the dicrotic notch and the ECG T-wave, reaching it lowest point at end diastole.

Pressure waveform measurements from different sites of the arterial tree will have varying morphologies that depend on the physical properties of the vascular bed. As the arterial pressure wave travels from the central aorta to the peripheral arterioles, several changes occur, which result from an alteration in impedance and harmonic resonance. As a consequence, the distal arterial pressure wave has a higher and steeper systolic peak, a dicrotic notch that appears later, a diastolic wave that is more prominent, and a lower end diastolic pressure than that seen in the aorta. This phenomenon is called distal pulse amplification and causes systolic blood pressure measured through the radial artery to be higher than the aortic pressure.[14] In contrast, the mean arterial pressure (MAP)

in the radial artery more closely approximates the mean aortic pressure, and therefore, clinical therapy is often better guided using the radial artery MAP rather than the radial artery systolic blood pressure.

When the pressure wave reaches the arteriolar bed in the periphery, there is a dramatic increase in vascular resistance, causing pressure wave reflection and further augmentation of the upstream arterial pressure pulse. The magnitude of this effect influences the shape of the arterial pulse wave in health and disease. For example, the arterial waveform pattern in the elderly patient differs from that seen in the young individual because reduced arterial distensibility causes early pressure wave reflection to augment the systolic pressure peak rather than the diastolic pressure wave (Fig. 5).[15]

Several disease states have characteristic arterial pressure waveforms. Pulsus alternans (congestive heart failure), pulsus bisferiens (aortic insufficiency), pulsus parvus and tardus (aortic stenosis), pulsus paradoxus (cardiac tamponade), and the "spike-and-dome" pattern seen in hypertrophic cardiomyopathy can be identified from direct recordings of the systemic arterial pressure.[5] These abnormal arterial pressure waveforms, although characteristic of underlying cardiovascular pathology, are not absolutely diagnostic. Accurate clinical interpretation depends on attention to the previously described technical details of arterial pressure measurement and requires that the clinical context be considered.

Influence of the Respiratory Cycle on the Arterial Pressure Waveform

One additional application of arterial pressure monitoring is the assessment of cardiac preload through analysis of the cyclic variation in arterial pressure that occurs during positive pressure mechanical ventilation. These variations demonstrate cardiopulmonary interactions that result from changes in intrathoracic pressure and lung volume. These can be described briefly as follows.

With the onset of positive pressure inspiration, the rise in lung volume effectively "squeezes" the pulmonary venous bed, increases drainage of blood from the pulmonary veins into the left atrium, and augments left ventricular preload. Concurrently, left ventricular afterload falls because of the increased intrathoracic pressure. The increase in left ventricular

FIGURE 5. *Early pressure wave reflection augments the systolic blood pressure in the elderly patient (right trace) compared with the younger patient (left trace).*

preload and decrease in afterload produce an increase in left ventricular stroke volume and an increase in systemic arterial pressure. At the same time, the increase in intrathoracic pressure seen in early inspiration reduces systemic venous return and decreases right ventricular preload and right ventricular stroke volume.

Toward the end of inspiration or during early expiration, the reduced right ventricular stroke volume that occurred during inspiration leads to reduced left ventricular filling, and as a result, left ventricular stroke volume falls and systemic arterial blood pressure decreases. This cyclic variation in systemic arterial pressure can be measured and quantified as the systolic pressure variation (SPV).

Systolic pressure variation can be further subdivided into inspiratory and expiratory components by measuring the increase (Δup) and decrease (Δdown) in systolic pressure as compared with the end-expiratory, apneic baseline pressure (Fig. 6). Normally a mechanically ventilated patient will have a Δup and Δdown of approximately 5 mm Hg each and a SPV of less than 10 mm Hg. In animals and humans, increased values of SPV and particularly Δdown have been shown to be extremely sensitive indicators of hypovolemia and responsiveness to fluid administration.[16,17] Exaggerated SPV remains present in many patients when compensatory mechanisms still maintain arterial blood pressure and central filling pressures values near normal yet hypovolemia is still present.[18]

Systolic pressure variation seen in clinical practice must be interpreted with caution. Although there is a cyclic change in arterial pressure during spontaneous ventilation, using SPV to predict hypovolemia has only been well validated in patients receiving positive pressure mechanical ventilation. The magnitude of the SPV will be influenced by ventilatory parameters, including tidal volume and peak inspiratory pressure. Values will be confounded further by the presence of arrhythmias, or changes in chest

Figure 6. *Systolic pressure variation (SPV) of the arterial blood pressure during positive pressure mechanical ventilation. Increased SPV, particularly increased Δdown, indicates hypovolemia.*

wall and lung compliance, addition of positive end expiratory pressure, and other pulmonary pathology.[19]

Indications for Central Venous Pressure Monitoring

Central venous pressure provides an indication of right heart preload and is influenced by intravascular volume, venous tone, and right ventricular function. Central venous pressure is considered equivalent to right atrial pressure, which, in the absence of tricuspid valve stenosis, is equal to right ventricular end diastolic pressure. In high-risk surgical patients, it is often important to measure CVP directly, because assessment of preload by physical examination is unreliable.[20] Of equal importance, central venous catheterization allows a variety of therapeutic options that are often needed in these patients, including administration of vasoactive drugs, providing access for pulmonary artery catheterization or ventricular pacing, and securing prolonged venous access for nutritional, antibiotic, or other pharmacologic support.

Complications of Central Venous Pressure Monitoring

Although adverse consequences from direct arterial catheterization are uncommon, the incidence of complications from central venous catheters is over 15%.[21] These can be categorized into mechanical, infectious, and thrombotic complications[22] (Table 1). Most, but not all, mechanical complications occur during catheter placement. Less commonly, delayed vascular perforation can cause serious bleeding, hemothorax, hemome-

Table 1. *Complications of Central Venous Pressure Monitoring*

Mechanical complications
Arterial puncture and cannulation
Hematoma
Hemothorax
Surrounding nerve injuries
Pneumothorax
Embolization of broken catheter or guide wire
Air embolism
Arrhythmias
Lymphatic system injury
Infectious complications
Sepsis
Endocarditis
Thrombotic complications
Venous thrombosis
Pulmonary embolism

diastinum, or cardiac tamponade. To diminish the risk of vascular perforation with catheter insertion in the left internal jugular or subclavian vein, the tip of the catheter should not be positioned against the wall of the superior vena cava.

Some common mechanical complications, including pneumothorax and hemothorax, are more likely with catheter insertion in the subclavian vein than in the internal jugular vein. Overall, however, vascular injuries are the most frequent complications of CVP placement. Unintended carotid artery puncture leading to hematoma formation and potential airway compromise is the most common major complication of internal jugular vein catheterization. The preferred site for emergency central venous access is the femoral vein, particularly during cardiopulmonary resuscitation. Like with most medical procedures, mechanical complications are lower when CVP cannulation is performed by more experienced physicians.[23]

The risks of mechanical complications can be reduced with the use of ultrasound guidance. This imaging technique can be used to identify the underlying vascular anatomy before attempted venipuncture, or real-time ultrasound guidance can be used using specially designed equipment that allows imaging during needle placement (Fig. 7). Real-time ultrasound imaging has been shown to decrease complications during internal jugular and femoral vein cannulation.[24] When central venous cannulation is
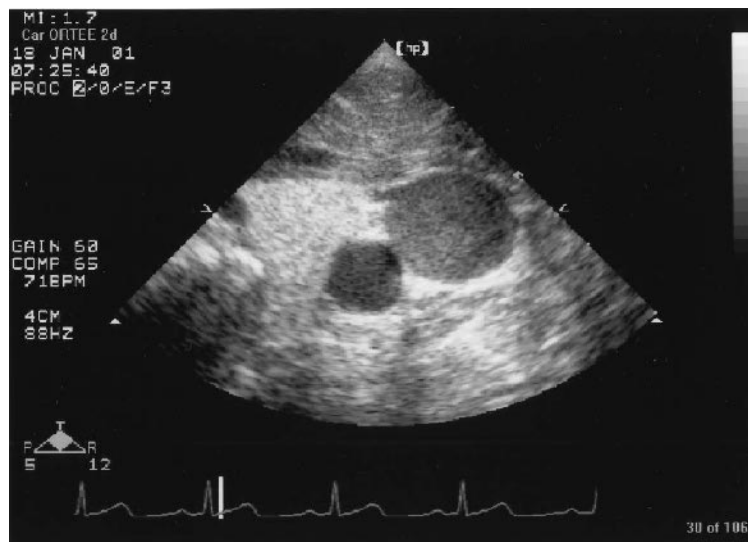

Figure 7. *Cross-sectional ultrasound image of the right internal jugular vein and its typical anatomic relation with the right common carotid artery. Note that the medial (left) aspect of the vein overlies the artery.*

performed immediately before surgery, it is probably safer to use the internal jugular vein, because placement in the subclavian vein carries a higher risk of pneumothorax, which could be difficult to diagnose intraoperatively and is more likely to become problematic during positive pressure mechanical ventilation.

To eliminate some of the mechanical complications of CVP monitoring, a catheter can be placed peripherally and used to estimate the CVP. Although measurements recorded from peripherally placed venous catheters show good agreement with centrally measured pressures under certain clinical conditions,[25,26] it is unlikely that the CVP obtained peripherally is suitably accurate under a wide variety of clinical situations.

Central venous catheters are a major source of infection in the intensive-care unit. Site of insertion has a significant influence on the rate of this complication. Subclavian vein catheterization is associated with a lower rate of infection than femoral or internal jugular vein catheterization.[21] Use of antibiotic-impregnated catheters significantly lowers the rate of catheter-related infections.[27,28] Because the risk of catheter-related infection is strongly influenced by the duration of catheter residence, these antibiotic-impregnated CVP catheters should be considered for patients who are expected to need the catheter to remain in place for more than 3 to 4 days. Indiscriminate use of these catheters should be avoided, because this could contribute to the emergence of antibiotic-resistant infections.

Catheter-related venous thrombosis is a significant problem in critically ill patients.[29] Its incidence varies with insertion site, and it has been reported to be as high as 21.5% with femoral catheter insertion, intermediate with internal jugular catheters, and as low as 1.9% with subclavian catheterization.[21,30]

Central Venous Pressure Waveform Morphology

The normal CVP waveform consists of *a*, *c*, and *v* waves, a systolic *x* descent, and a diastolic *y* descent. The individual waveform components have a predictable relation to the electrocardiogram (Fig. 8). The *a* and *c* waves result from atrial and ventricular contractions, respectively, whereas the *v* wave results from right atrial filling driven by systemic venous return. For clinical purposes, it is best to consider the normal CVP to have 3 systolic components (*c* wave, *x* descent, *v* wave) and 2 diastolic components (*y* descent, *a* wave).

The characteristics and amplitude of the CVP waveform components change significantly with arrhythmias and tricuspid valve pathology. During junctional rhythm, atrial contraction will occur late when the tricuspid valve is already closed, owing to delayed retrograde atrial depolarization. A tall *a* wave, termed a cannon *a* wave, is observed, and its timing and

Figure 8. *Normal central venous pressure waveform and its relation to the electrocardiogram. Note the 3 systolic components (*c *wave,* x *descent,* v *wave) and 2 diastolic components (*y *descent,* a *wave).*

amplitude vary according to the precise timing of right atrial contraction (Fig. 9). Because the "atrial kick" is lost during junctional rhythm, stroke volume, cardiac output, and systemic blood pressure will be markedly decreased in patients dependent on this booster pump for adequate ventricular filling. In patients with atrial fibrillation, the CVP *a* wave is absent and, like in junctional rhythm, ventricular filling can be impaired.

Tricuspid stenosis produces a CVP that displays a prominent *a* wave and an attenuated *y* descent that reflect the obstruction to right atrial-emptying. A tall *a* wave can also be present in patients with right ventricular diastolic dysfunction. With tricuspid regurgitation, retrograde filling of the right atrium causes the *c* and *v* waves to merge, eliminating the *x* descent and resulting in a large holosystolic regurgitant *cv* wave (Fig. 9). It is important to recognize that the digital value for CVP displayed on the electronic monitor will be influenced by these tall *a* or *v* waves and not necessarily reflect the end diastolic pressure indicative of right ventricular preload.

Other less common conditions can have a striking influence on the shape of the CVP waveform. In pericardial constriction, the mean CVP is increased, the *a* and *v* waves are prominent, and the *x* and *y* descents are
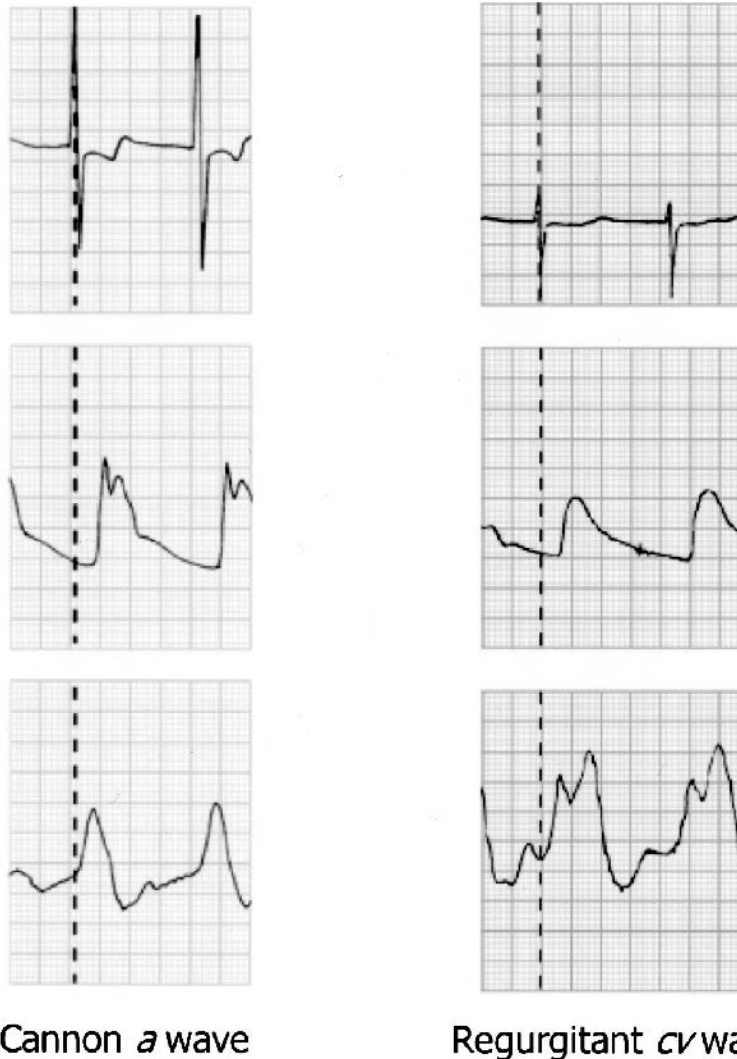
Cannon *a* wave

Regurgitant *cv* wave

Figure 9. *Central venous pressure (CVP) waveforms in junctional rhythm (left) and tricuspid regurgitation (right). The electrocardiogram (top), arterial blood pressure (middle), and CVP (bottom) traces are shown. Note that a tall systolic wave is seen in both cases. In junctional rhythm, this is a cannon* a *wave, and in tricuspid regurgitation, this is a regurgitant* cv *wave, the latter being a longer duration holosystolic wave.*

steep (Fig. 10). In contrast, cardiac tamponade also results in a marked elevation of CVP, but the *y* descent is characteristically attenuated. Note that the increased CVP seen in both constriction and tamponade reflects the extramural compressive effect of the restrictive pericardium or effusion, and cardiac filling is reduced in both conditions.
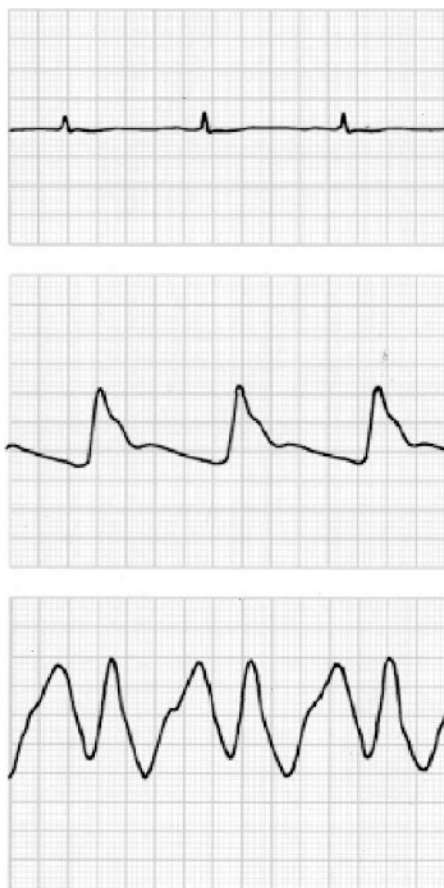
FIGURE 10. *Pericardial constriction produces a central venous pressure trace (bottom) that displays tall* a *and* v *waves and steep systolic* x *and diastolic* y *descents. The electrocardiogram (top) and arterial blood pressure (middle) traces are also shown.*

Influence of the Respiratory Cycle on the Central Venous Pressure Waveform

Although normal cardiopulmonary interactions afford the opportunity to use direct arterial pressure monitoring to assess the adequacy of intravascular volume, these same interactions often lead to misinterpretation of central vascular pressure measurements, including pulmonary artery wedge pressure and CVP. It is important to note the critical difference between measured central vascular pressures and the more physiologically relevant transmural pressures. Measured or transduced central vascular pressures are referenced to ambient atmospheric pressure, whereas transmural pressures reflect the difference between the transduced pressure inside the cardiac chamber and the surrounding extramural pressure in the pericardium or thorax. Changes in intrathoracic pressure that occur during the respiratory cycle generally influence the measured central vascular pressures in one direction, whereas simulta-

neously, the transmural pressure changes in the opposite direction. For example, during spontaneous respiration, measured CVP decreases with the decrease in intrathoracic pressure during inspiration (Fig. 11). Although the transduced CVP will be reduced, the lower CVP promotes increased venous return and the transmural CVP actually increases.

The opposite pattern is observed during positive pressure mechanical ventilation (Fig. 11). During inspiration, intrathoracic pressure increases and the measured CVP also increases, whereas transmural CVP actually decreases. The lower transmural CVP reflects reduced right atrial filling and the known reduction of cardiac preload that occurs during positive pressure ventilation. Like in the case of spontaneous ventilation, the change in measured CVP during inspiration is in the opposite direction to the change in transmural CVP.
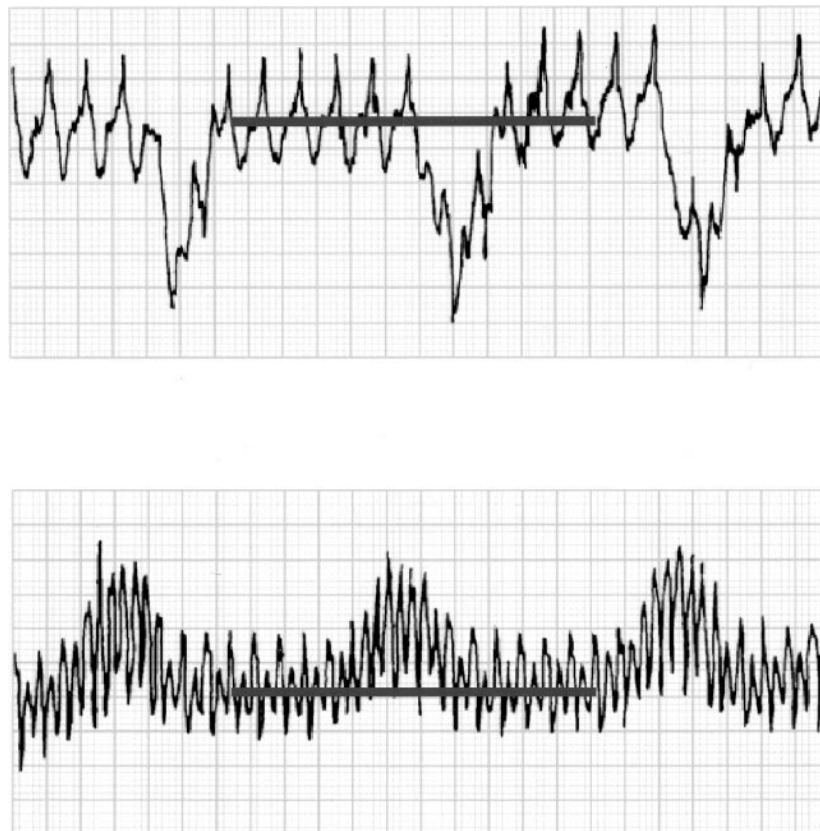

Figure 11. *Respiratory variation in central venous pressure (CVP). During spontaneous inspiration, measured CVP decreases (top). During positive pressure inspiration, measured CVP increases (bottom). The CVP should be recorded at end expiration (before onset of inspiration, dark horizontal lines) to avoid the confounding effects of changing intrathoracic pressure.*

As a consequence of these confounding changes in intrathoracic pressure that occur during the respiratory cycle, interpretation of measured or transduced CVP values is complicated. Because the goal of CVP measurement is to assess right heart preload, it is desirable to eliminate these confounding respiratory effects. To accomplish this, CVP measurements always should be made at end expiration, when there are minimal effects from changes in intrathoracic pressure that occur during either spontaneous or positive pressure inspiration (Fig. 11).

It is also important to remember a single measurement of CVP might not be adequate to estimate preload, because CVP is influenced by venous tone, intravascular volume, and right ventricular function. The CVP trend provides a better estimation of preload. In addition, response to a volume challenge of 250 to 500 mL will provide an indication of volume-responsiveness of the patient under the existing clinical conditions.

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