Monitoring With CVP and PAC

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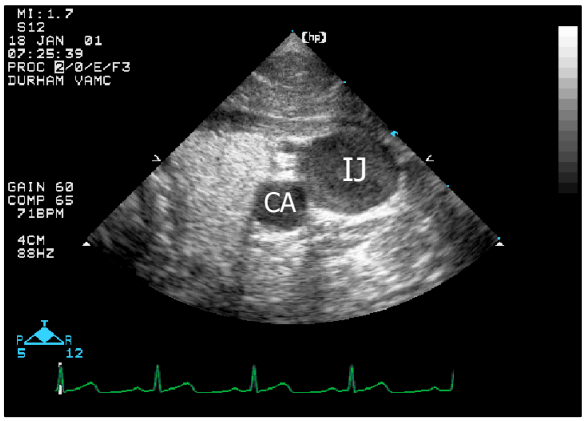
Complications of Central Venous Catheterization

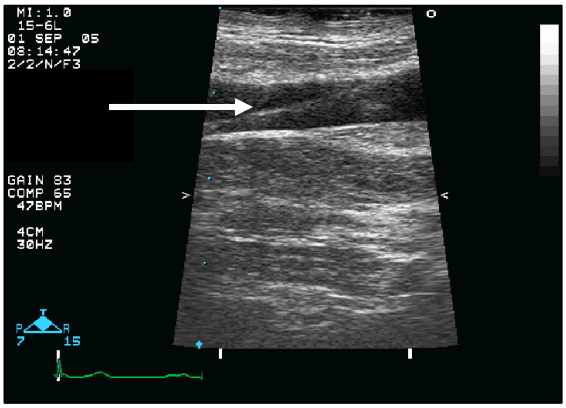
Central venous catheterization (CVC) remains a common procedure for the care of intensive care and high-risk surgical patients. The complications are well recognized, and the more common include:

1. Bleeding (adjacent arterial injury, hematoma formation, airway compromise, or cardiac tamponade)
2. Pneumothorax, hemothorax, and chylothorax
3. Nerve injury
4. Infection (bacteremia, sepsis, endocarditis)
5. Venous thromboembolism
6. Venous (and paradoxical) air embolism

Numerous other complications have been reported, and a recent ASA Closed Claims Analysis (Domino 2004) suggested that nearly 2% of claims in the database pertain to CVC-associated injuries. Compared to other claims in this database, CVC claims were associated with a higher average severity of injury and increased fatality. The most common claim related to catheter or wire embolism, undoubtedly the result of a medical error, and other common complications described were as noted above. Perhaps of greatest importance, the authors determined that nearly half of these injuries were preventable by use of pressure waveform monitoring, chest radiography, or ultrasound-guided cannulation. Growing use of ultrasound for CVC is based on clinical evidence of reduced failure rates and complications compared to standard techniques based on surface anatomy and palpation (Randolph 1996). Inexpensive ultrasound devices are widely available, easy to use, and should be considered routinely for all difficult cannulation procedures. For example, the easily compressible internal jugular vein can is rapidly identified and distinguished from the adjacent carotid artery, and successful placement of the guidewire can be confirmed.



Transverse ultrasound image of right internal jugular (IJ) vein lying slightly lateral and superficial to the right carotid artery (CA).



Long axis ultrasound image of right internal jugular vein showing the guidewire in the lumen (arrow).

Pulmonary artery catheterization carries additional risks beyond those associated with central venous catheterization, including risks associated with catheter flotation through the right heart chambers and prolonged catheter residence in the pulmonary artery (Roizen, ASA Task Force on PAC 2003). During passage of a pulmonary artery catheter (PAC) through the right heart chambers, non-sustained atrial and ventricular dysrhythmias are common and of no clinical significance. Less frequent but more life threatening complications include right bundle branch block leading to complete heart block in patients with pre-existing left bundle branch block or sustained ventricular dysrhythmias, including ventricular fibrillation, particularly in patients with myocardial ischemia involving the right ventricle (Lopez-Sendon 1990). Catheter residence within the pulmonary artery for hours or days can be complicated by infection (endocarditis and sepsis), pulmonary infarction, and pulmonary artery rupture, conditions which are often fatal.

The infectious risks of both PAC and CVC placement can be reduced by adherence to practice guidelines for catheter placement promulgated by JCAHO and the Institute for Healthcare Improvement (IHI) (Berwick 2006). The IHI 100,000 Lives Campaign has identified six areas for improving patient care, including prevention of CVC-related infection by proper hand hygiene, maximal barrier precaution (including a large patient drape), chlorhexidine skin preparation, optimal catheter site selection, and prompt removal of unnecessary catheters. As anesthesiologists, we need to be concerned with complications that may result from our care but not clinically manifest for days after surgery.

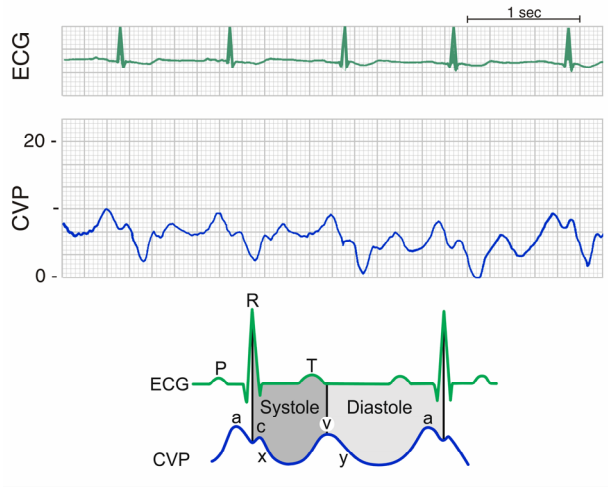
Technical Considerations for Measuring Intravascular Pressures

Prior to initiating pressure monitoring, the transducers must be "zeroed" and "leveled." While often done at the same time, these are distinct procedures, both of which are required for accurate pressure measurement. Zeroing refers to the procedure in which the transducer is exposed to ambient atmospheric pressure through an attached stopcock open to air, and the atmospheric pressure is assigned a pressure of 0 mmHg when the monitor zero button is pushed. Note that this means that all intravascular pressure measurements are referenced to ambient atmospheric pressure.

The second step in transducer setup is to determine the proper external reference level for the pressure transducer, or leveling the transducer. Although the mid-chest position is chosen commonly, this level has no consistent relation to the position of the heart (or right atrium) within the thorax. Positioning the transducer in this location may produce a significant hydrostatic error, which causes overestimation of central vascular pressures by more than 5 mmHg in the average patient. Cogently summarized, "the only factor that contributes to measured hydrostatic pressure with a fluid-filled catheter system is the position of the transducer with respect to the uppermost fluid level in the … chamber in which pressure is being measured." (Courtois 1995) It follows that the preferred position for an external transducer is the top of the right atrium, which is estimated best at a point approximately 5-cm below the sternal border at the 4th intercostal space. Transducers for measuring central venous pressure (CVP) and pulmonary artery pressure (PAP) should be placed in this position.

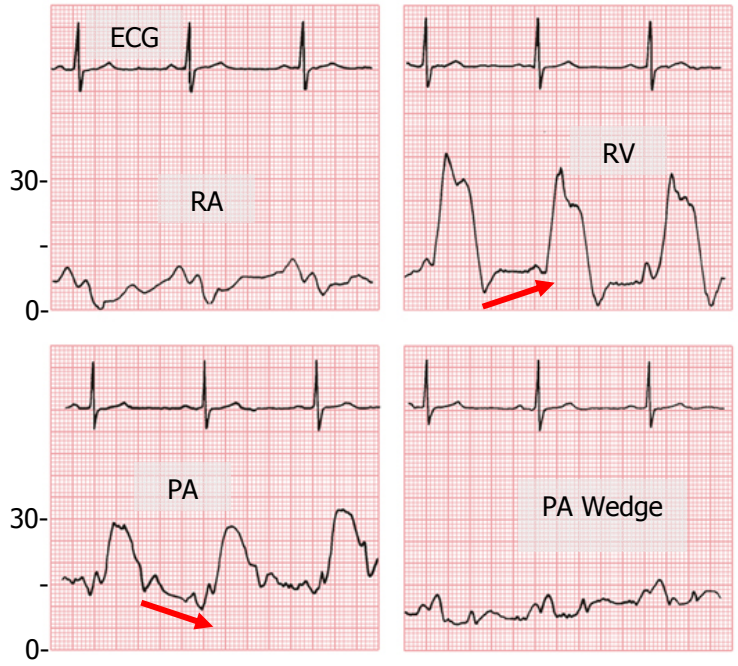
Physiologic Basis for CVP and PAC Waveforms and Interpretation of Cardiac Filling Pressures

The normal CVP waveform consists of three peaks (a, c, v) and two descents (x, y). The most prominent wave is the **a wave**, which results from atrial contraction following the ECG P wave at end-diastole. This atrial contraction provides the end-diastolic atrial kick, which loads the right ventricle through the open tricuspid valve. Atrial pressure decreases following the a wave, as the atrium relaxes. This decline in atrial pressure is interrupted by the **c wave** at the beginning of systole. The c wave results from isovolumic right ventricular contraction, which closes the tricuspid valve, causing it to bow back toward the right atrium in early systole and produce an increase in atrial pressure. The c wave must follow onset of the ECG QRS, since it is an early systolic event. Atrial pressure continues its decline in mid-systole, owing to ongoing atrial relaxation and changing atrial geometry produced by ventricular contraction and ejection. This is the **x descent** or systolic collapse in atrial pressure. The x descent can be divided into x and x' descents, denoting the portions before and after the c wave. However, the x descent generally should be considered to be the systolic decline in atrial pressure. The last atrial pressure peak is the **v wave**, caused by venous filling of the right atrium, through the vena cava, during late systole while the tricuspid valve remains closed. The v wave peaks just after the ECG T wave. Atrial pressure then decreases as the tricuspid valve opens and blood flows from atrium to ventricle. This is the **y descent** or diastolic collapse in atrial pressure.

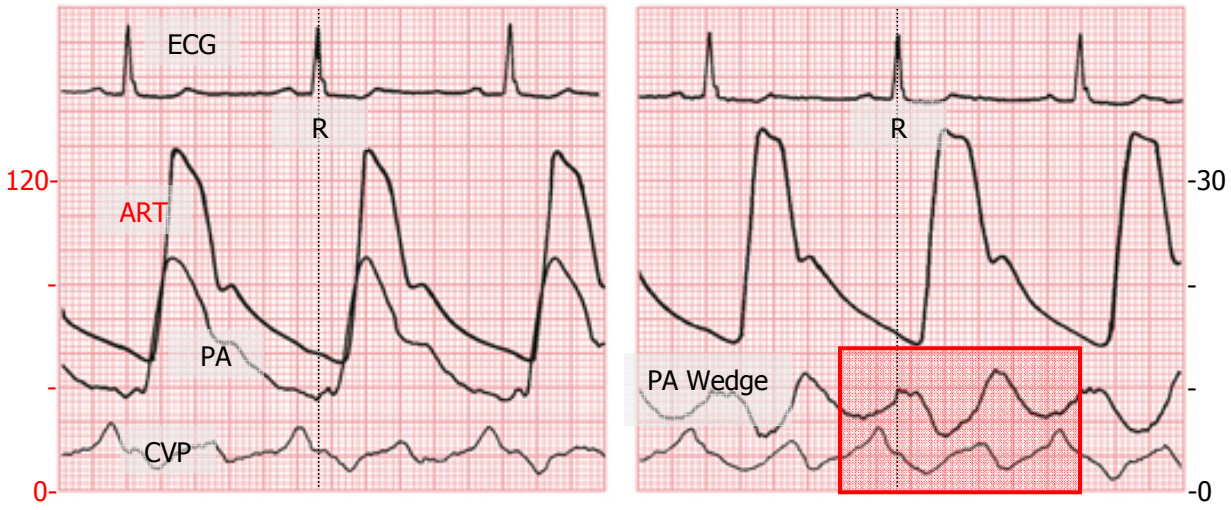


During flotation of a PAC through the right heart chambers, typical pressure waveforms are recorded from the right atrium, right ventricle, and pulmonary artery. Note that right atrial (RA) pressure is equal to CVP and of similar waveform morphology. As the PAC crosses the tricuspid valve, the increase in systolic pressure identifies

arrival of the catheter in the right ventricle (RV), and flotation across the pulmonic valve is identified by an increase in diastolic pressure in the pulmonary artery (PA). Occasionally, distinguishing RV from PA pressure is difficult, but careful examination of the diastolic portion of these two pressure waveforms clarifies these different catheter tip locations. During diastole, RV filling results in a pressure increase in the RV, while diastolic flow from the PA toward the lung results in a pressure decrease (Red Arrows).

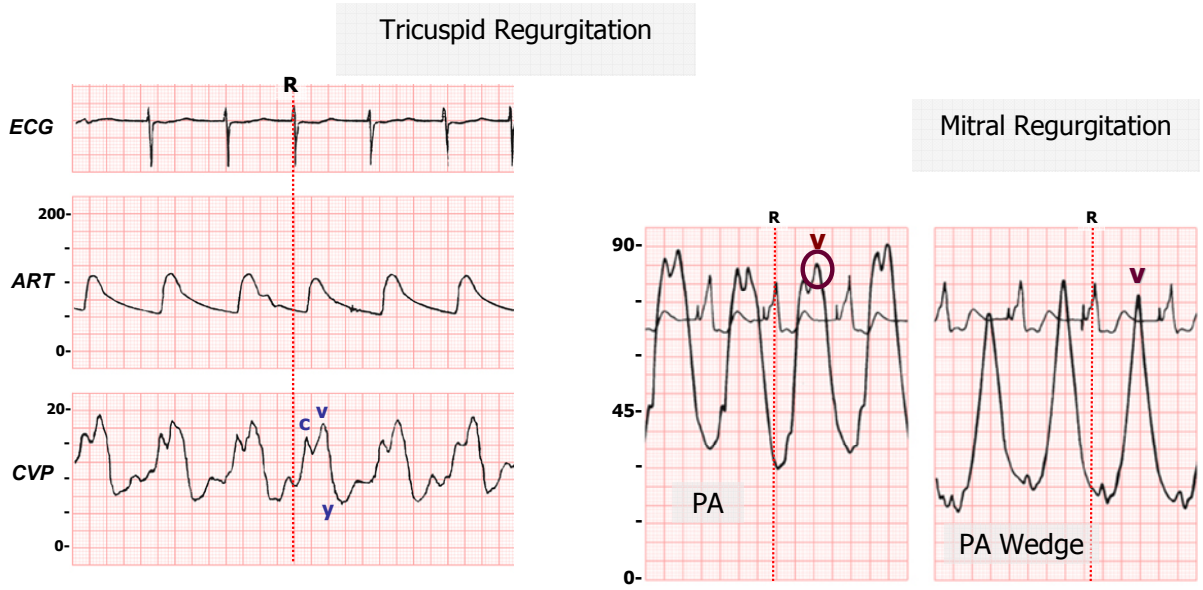


Advancing the balloon tipped PAC further into the PA will allow the catheter to "wedge" and record the PA wedge pressure or PA occlusion pressure. When the PAC is wedged, the pressure recorded from the catheter tip is isolated from the proximal PA pressure by the occluding balloon, and the tip records downstream pulmonary venous and left atrial pressure. Normal PA wedge pressure waveforms display the same characteristic peaks and descents previously described for CVP or RA pressure waveforms. However, PA wedge pressure is an *indirect* measure of left atrial pressure. Left atrial pressure waves (a, c, and v) must be transmitted in a retrograde fashion, from the left atrium, through the pulmonary veins, capillaries, and arteries to arrive at the wedged PAC. As a result, ***PA wedge pressure is a delayed, damped reflection of left atrial pressure***. Left atrial pressure a and v waves are recorded in a pulmonary artery wedge pressure trace approximately 150-200 msec after they are inscribed in the left atrial pressure trace, largely owing to the transmission time through the interposed pulmonary vascular bed. In general, the wedge pressure a wave follows the ECG R wave owing to this delay (compare CVP and PA wedge pressures in red box). If these a and v waves are prominent, they can be detected in the unwedged PA pressure trace, the a wave distorting the systolic upstroke, and the v wave obscuring the dicrotic notch.



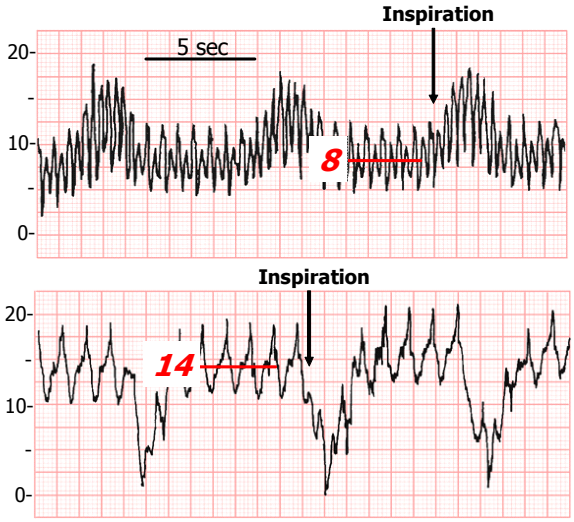
A wide variety of hemodynamic abnormalities can be identified through careful analysis of CVP and PAC waveforms. Dysrhythmias have characteristic signatures. In **atrial fibrillation** the CVP a wave disappears, and the c wave becomes more prominent, since atrial volume is greater at end-diastole and onset of systole, owing to the absence of atrial contraction. **Isorhythmic atrioventricular dissociation** or **junctional rhythm** alters the normal

sequence of atrial and ventricular contraction. When there is retrograde conduction of the pacemaker impulse from AV node to the atrium, atrial contraction occurs during ventricular systole, when the tricuspid valve is closed, thereby inscribing a tall cannon a wave in the CVP waveform. Absence of normal atrioventricular synchrony during ventricular pacing can be identified in similar fashion by searching for venous cannon waves. In these instances, the CVP helps diagnosis the cause of arterial hypotension since loss of the normal end-diastolic ECG P wave may not be as clearly evident as the cannon a waves in the CVP trace. Valvular heart disease may also be identified, particularly AV valve regurgitation. **Tricuspid regurgitation** produces abnormal systolic filling of the right atrium through the incompetent valve. A broad, tall systolic c-v wave is inscribed, which begins in early systole and obliterates the systolic x descent in atrial pressure. The CVP trace is said to be ventricularized, resembling RV pressure. This regurgitant wave differs in onset, duration and magnitude from a normal CVP v wave caused by end-systolic atrial filling from the vena cavae. Right ventricular end-diastolic pressure is overestimated by the numeric readout on the bedside monitor, which reports a single mean value for CVP. Instead, right ventricular end-diastolic pressure is estimated best by measuring the CVP value at the time of the ECG R wave, prior to the regurgitant systolic wave. In a similar manner, a tall systolic c-v wave noted in the PA wedge pressure trace is the typical hallmark of severe **mitral regurgitation**. As noted above, a tall v can be noted to distort the PA waveform in the unwedged PA pressure trace.



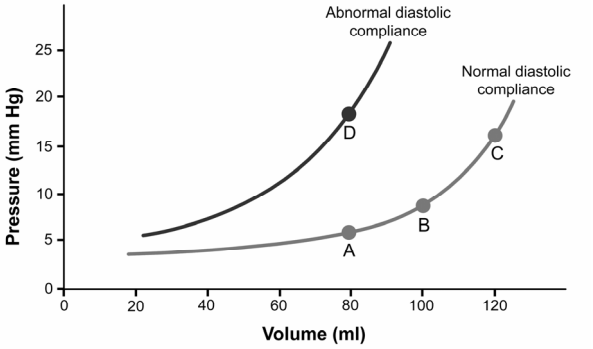
Perhaps the most clinically important pressure waveform abnormalities are those introduced during the respiratory cycle and caused by cyclic variations in intrathoracic pressure. During spontaneous breathing, inspiration causes a decrease in pleural and pericardial pressures that is transmitted to the RA and produces a decline in transduced CVP (as well as other measured central vascular pressures). Note that the decrease in the transduced CVP measured during inspiration may actually reflect an increase in transmural CVP, which is calculated as the difference between the pressure measured inside the RA and pericardial pressure outside the atrium. In clinical practice, transmural pressures are rarely determined owing to the difficulties in assessing pleural or pericardial pressures. Instead, **end-expiratory pressure** should always be recorded to provide the best estimate for transmural filling pressures. Since pleural and pericardial pressures approach atmospheric pressure at end-expiration whether the patient is breathing spontaneously or receiving positive pressure mechanical ventilation, pressures recorded at this point in the respiratory cycle will provide the best estimate for transmural pressure and cardiac preload. End-expiratory pressure values can be determined by visual inspection of the CVP, PA, and wedge waveforms on a calibrated monitor screen or paper recording.

Measurement of CVP at End-Expiration



Positive Pressure Ventilation

Spontaneous Ventilation



An understanding of transmural pressure is the key to proper interpretation of intravascular pressure recordings and their relationship to cardiac filling volume or preload. Increased filling pressure can result from increased chamber volume, increased chamber wall stiffness, or increased pressure surrounding the heart (pericardial or intrathoracic pressure). This is the physiologic basis for recording intravascular pressures at end-expiration and explains why high CVP or PA wedge pressures may not always indicate hypervolemia or increased chamber volume. For example, when there is diastolic left ventricular dysfunction from myocardial ischemia (impaired relaxation) or hypertrophy (increased wall stiffness), the shape of the ventricular pressure-volume relationship is shifted up and to the left. Under normal conditions, a fluid challenge would increase ventricular preload with little change in filling pressure (Point A → Point B). Owing to the curvilinear relationship between chamber pressure and volume, marked increases in chamber filling will be accompanied by an increased filling pressure (Point C). On the other hand, when there is diastolic dysfunction, the same elevated filling pressure may not reflect increased preload, but rather abnormal chamber stiffness or increased surrounding pressure (Point D). It is likely that these physiologic considerations confound simple interpretation of PAC-derived data and contribute to the limited value of PAC monitoring found in many clinical trials.

Pulmonary Artery Catheterization and Patient Outcome

The PAC has been surrounded by controversy since its introduction into clinical medicine by Swan and Ganz nearly 40 years ago (Swan 1970). A number of early clinical trials identified excess mortality in patients receiving PAC monitoring, but these studies were criticized for their retrospective design and other shortcomings. The SUPPORT Trial was perhaps the most widely publicized of these studies, as it was a large trial conducted in critically ill medical and surgical patients from five major medical centers. The patients with PAC monitoring were carefully matched to other ICU patients through use of a propensity score, but still had increased hospital length of stay, increased hospital costs, and higher mortality than patients who were not monitored with PACs (Connors 1996). Widespread use of these catheters continued, in part owing to concerns that randomized prospective clinical trials were still not available. However, several studies in the past few years have provided new and important data.

Sandham et. al. randomized PAC usage in nearly 2,000 ASA III and IV patients undergoing urgent or elective major surgery and found identical survival in patients with and without PAC monitoring (Sandham 2003). Although the authors did not identify excess mortality associated with PAC usage, there were more pulmonary complications in the PAC group, including pulmonary embolism, hemorrhage, and infarction. Another seminal

randomized prospective investigation was conducted in 1,041 patients from 65 intensive care units in the United Kingdom. This PAC-Man Trial showed no difference in hospital mortality in patients monitored with PACs compared with control patients (Harvey 2005). A third randomized prospective study, the ESCAPE Trial, evaluated PAC use in hospitalized patients with severe congestive heart failure and found no effect on mortality or days alive out of hospital (ESCAPE 2005). And perhaps of greatest clinical impact, a meta-analysis of 13 randomized clinical trials conducted between 1985 and 2005 in 5,051 patients, half of whom were surgical, showed that PAC monitoring had NO EFFECT on mortality or days hospitalized (Shah 2005).

Taken together, these most recent publications suggest that we have little clinical evidence that PAC monitoring benefits our patients. Many have argued that clinicians, both doctors and nurses, who use PACs are inadequately trained in their use and have inadequate knowledge to use these devices safely (Iberti 1990). If we are to continue using this invasive monitoring tool, we must understand its limitations, the physiologic principles underlying interpretation of the data provided, and recognize the potential associated complications before they harm patients.

All figures modified courtesy of Jonathan B. Mark, Atlas of Cardiovascular Monitoring, 1998 (Churchill Livingstone)

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