

## Central Venous Pressure and the Pulmonary Artery Catheter

**Dr Frank Schneider**

*UCT Dept of Anaesthesia & Perioperative Medicine*

Central venous pressure (CVP) and pulmonary artery pressure (PAP), used as an indication of cardiovascular filling, have been long-standing components of haemodynamic monitoring and optimisation. Both central venous catheters (CVC) and pulmonary artery catheters (PAC) require central vein access, with the inherent risks and complications associated with this invasive procedure. Additional complications related to transcatheter passage and the pulmonary vasculature are relevant to PAC use. The following notes will summarise the important aspects of CVP and PAP monitoring in routine clinical use.

### Central Venous Pressure

This is the pressure within the intrathoracic vena cava, is usually measured by the insertion of a CVC in the internal jugular or subclavian vein, and equates to right atrial pressure in the absence of caval obstruction. The point of reference and zeroing of a transducer is at the level of the right atrium, at the 4<sup>th</sup> intercostal space in the mid-axillary line ("phlebostatic axis" when supine). By convention, pressure should be recorded at the base of the c-wave, at end expiration in the supine position, which equates to right atrial pressure just prior to ventricular systole.

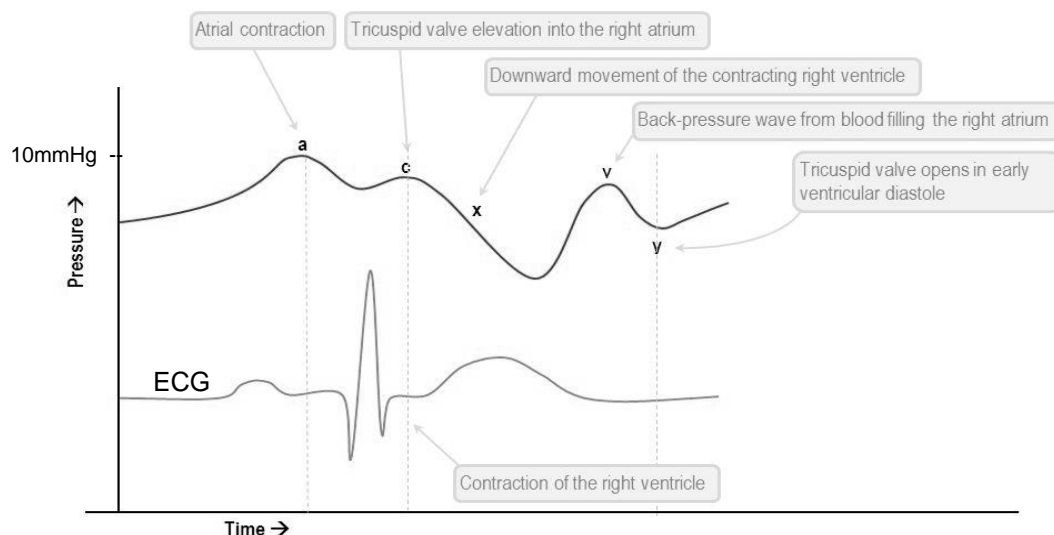
The normal CVP waveform is divided into:

**'a' wave** – caused by increased right atrial pressure during atrial contraction and correlates with the P wave on the ECG. It disappears in atrial fibrillation or may be seen as flutter waves in atrial flutter. The descent of the 'a' wave occurs with atrial relaxation and is interrupted by the 'c' wave.

**'c' wave** – closure of the cusps of the tricuspid valve occurs as right ventricular pressure rises during isovolumetric contraction of the right ventricle, at the end of the QRS complex. Right atrial pressure rises due to the cessation of blood flow across the tricuspid valve.

**'x' descent** – as the right ventricle contracts and the tricuspid annulus moves towards the cardiac apex (TAPSE – tricuspid annular plane systolic excursion, cf. as seen on echo), the right atrium is stretched and right atrial pressure decreases. This occurs before the T wave on ECG.

Figure 1 - Right atrial pressure waveform (adapted from [www.derangedphysiology.com](http://www.derangedphysiology.com))

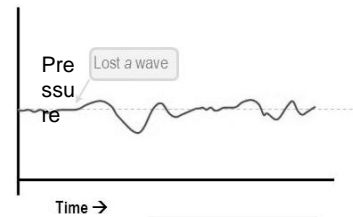


**'v' wave** – reflects rise in atrial pressure with continued blood inflow against a closed tricuspid valve throughout late systole and early diastole (after the T wave on ECG) and distension of the right atrium.

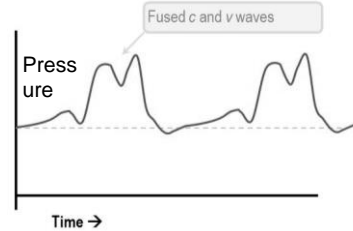
**'y' descent** – the tricuspid valve opens as soon as right atrial pressure exceeds right ventricular pressure, causing a rapid drop in pressure as atrial blood flows into the right ventricle, just before the P wave on the ECG. The third heart sound (S3) corresponds to the nadir of the 'y' descent, with the subsequent ascent reflective of continued atrial filling.

Abnormal CVP waveform patterns can be seen in the following circumstances:

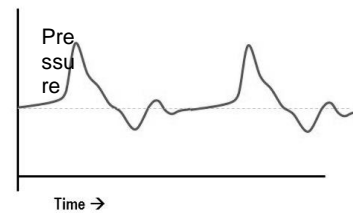
**Atrial fibrillation (AF) or flutter.** As the 'a' wave is caused by atrial contraction, it disappears during atrial fibrillation, or may be appreciated as flutter waves in atrial flutter. Long-standing AF may lead to atrial dilatation and rise in right atrial pressure.



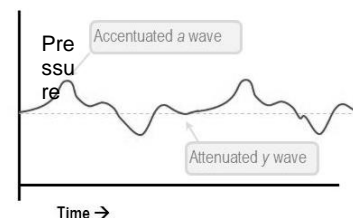
**Tricuspid regurgitation.** During right ventricular systole, blood is ejected through an incompetent tricuspid valve into the right atrium, causing fusion of the 'c' and 'v' waves, and obliteration of the 'x' descent (as it reverses the effect of TAPSE). Mild regurgitation may not affect the waveform pattern.



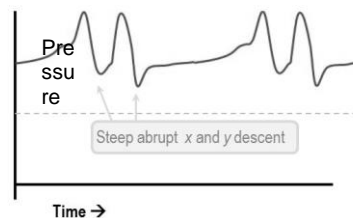
**Atrioventricular dyssynchrony.** Fusion of the 'a' and 'c' waves can occur during junctional rhythms, as atrial and ventricular contraction occur simultaneously. Cannon 'a' waves occur when atrial contraction coincides with a fully closed tricuspid valve, as seen during complete heart block, premature beats, or retrograde conduction of ventricular depolarization (e.g. ventricular tachycardia).



**Reduced right atrial emptying.** Increased resistance to right atrial emptying during atrial systole produces a large 'a' wave and attenuated 'y' wave. This may be due to flow obstruction at the tricuspid level (tricuspid stenosis, atrial myxoma or thrombus, carcinoid disease), or beyond (right ventricular hypertrophy, pulmonary stenosis, pulmonary hypertension).



**Pericardial constriction.** In constrictive pericarditis and restrictive cardiomyopathy the CVP is raised and a sharp 'y' descent, or bifid waveform with abrupt 'x' and 'y' descents may be seen. In cardiac tamponade, CVP is elevated, 'x' and 'y' descents are not prominent, and the 'y' descent is prolonged.



[www.derangedphysiology.com](http://www.derangedphysiology.com)

With a competent tricuspid valve, right atrial pressure (equal to CVP in the SVC) should equal right ventricular end-diastolic pressure and may predict preload when right ventricular compliance is normal. However, the variation in right ventricular compliance amongst many other factors such as intrinsic systolic and diastolic myocardial function, pulmonary vascular resistance, and positive pressure ventilation, has shown CVP to be a poor predictor of fluid responsiveness. At best, CVP should be used as a trend value and correlated with the individual clinical context.

## Pulmonary Artery Catheter

The Swan-Ganz catheter (or PAC), was introduced by Swan and Ganz in 1970 when they reported catheterization of the heart using their flow-directed balloon-tipped catheters. Their use in intensive care has decreased significantly in recent years, following multiple publications of lack of evidence for benefit or harm in critically ill patients, notably the PAC-Man trial in 2005. The development of less invasive cardiac output monitors has also contributed to the decline in PAC use, as has the marked increase in utilisation of echocardiography, which can provide most of the haemodynamic data available from the PAC. However, in complex patients with known pulmonary hypertension, severe left or right ventricular dysfunction, or severe valvular disease, the PAC may still be justified with echocardiography as a complementary technique.

Suggested indications for PAC placement might include:

- Investigation and quantification of cardiac shunts
- Perioperative monitoring: complex coronary and valvular surgery, vascular surgery, solid organ transplantation
- Critical care: severe left or right ventricular failure, pulmonary hypertension, constrictive pericarditis, valvular heart disease, obtaining mixed venous oxygen saturations, continuous cardiac output monitoring
- Therapeutic: drug delivery to pulmonary circulation (e.g. prostacyclin), cardiac pacing

Contraindications to insertion include prosthetic tricuspid or pulmonic valves, right-sided valvular vegetations or thrombi and intracardiac right mass.

Enthusiast centres continue to use PACs routinely, with good results and minimal complications. Furthermore, PACs are still the gold standard against which novel cardiac output monitors are compared. Emphasis should be put on using haemodynamic data, irrespective of how they are obtained, to inform therapeutic decision-making and appropriate patient management.

### Catheter

Modern adult PACs are 7 or 7.5 French gauge (Fr) and 110cm long, with 10 cm markings along the length to aid insertion, four or five lumens, and an inflatable 1.5ml balloon at the tip of the catheter. A blue lumen and proximal injectate port terminates at 30cm from the tip of the catheter, and should lie within the right atrium when the PAC is correctly sited. The white proximal infusion port terminates at 31cm from the tip, should also lie in the right atrium, and can be used for fluids and medication. The yellow distal port is the pulmonary artery lumen and allows measurement of pulmonary artery (PA) pressures and mixed venous saturation. A shorter red lumen connects to the balloon and allows inflation or deflation, with a locking mechanism to prevent deflation during flotation. A thermistor is located 4cm from the catheter tip, is attached to a proximal thermistor connector and used for thermodilution cardiac output measurement. Additional connectors may allow pacing capabilities, continuous cardiac output and oximetry measurement via fiberoptic sensors.

### Insertion

The catheter is inserted using a sterile Seldinger technique similar to CVC insertion, within a sterile cover (to allow manipulation of the PAC) via an 8 or 8.5Fr introducer sheath with a high volume sideport. The PA transducer is attached to the distal lumen to display waveform changes during flotation.

The catheter, in adults, is advanced 15-20cm from the jugular vein (10-15cm from subclavian, 30-40cm from femoral) to the right atrium. Following balloon inflation (usually max 1.5ml air), the catheter is advanced a further ~10cm, whilst watching for ectopic beats and a change to the right ventricular waveform. Systolic pressure is higher in the right ventricle (15-30mmHg) with diastolic pressure similar to right atrial pressure (1-6 mmHg).

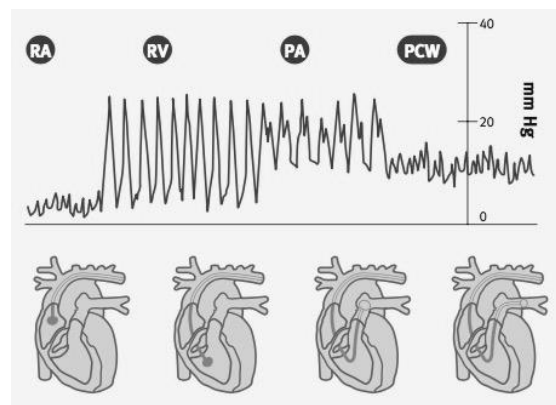


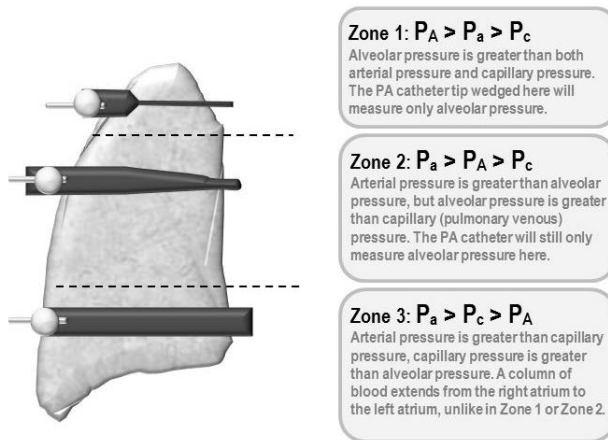
Figure 2 – Pressure waveform during advancement of PAC

The catheter should now be advanced a further ~10cm in order to float into the pulmonary artery. Pulmonary artery systolic pressure should be almost equivalent to right ventricular systolic pressure. Closure of the pulmonic valve causes a dicrotic notch on the pulmonary artery tracing, and special attention should be paid to the diastolic pressures, which are higher in the pulmonary artery. The catheter is advanced further until it is “wedged” in a central branch of the pulmonary artery, and now measures pulmonary artery wedge pressure (PAWP) or pulmonary artery occlusion pressure (PAOP), and the waveform shown will be that of left atrial pressures.

Insertion of the PAC may be hampered by tricuspid regurgitation or coiling in the right ventricle (RV). Transoesophageal echocardiography (TOE) allows real-time visualisation of the passage of the PAC from the right atrium, through the RV and into the PA (from a mid-oesophageal RV inflow-outflow view), and confirmation of the catheter tip in the pulmonary artery (>90% float into the right PA). Depending on the clinical setting, fluoroscopy or chest x-ray may also be used to confirm placement, as well as location within West Zone III.

### Pulmonary artery wedge pressure

This represents left atrial filling pressure (normally 6-12 mmHg) and in patients with normal physiology estimates left ventricular end-diastolic pressure (LVEDP), which gives an indication of left ventricular end-diastolic volume (LVEDV), but – like CVP – does not estimate preload or predict cardiac performance or fluid responsiveness. The catheter tip should be in West Zone III for accurate measurements, ensuring a static column of blood between the catheter tip in the PA and the left atrium. PAWP will be greater than LVEDP in mitral stenosis or regurgitation, with atrial myxoma, pulmonary venous obstruction (due to fibrosis or vasculitis), left-to-right shunt, COPD and positive pressure ventilation. PAWP will be less than LVEDP in left ventricular failure, raised intra-thoracic pressure, decreased left ventricular compliance and aortic regurgitation.



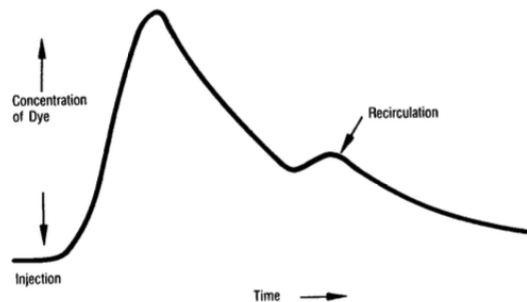
In addition to PAWP, the most common measured and derived parameters, with normal values, are as follows:

MEASURED VALUES	
Cardiac Output (CO)	4 – 8 l/min
Cardiac Index (CI)	2.5 – 4 l/min
CVP	2 – 6 mmHg
PAWP	6 – 12 mmHg
PA Pressure (PAP)	25/10 mm Hg
SvO <sub>2</sub> (mixed venous)	65 – 70%
Temperature	
DERIVED VALUES	
Stroke Volume (SV)	50 – 100 ml/beat
SV Index (SVI)	25 – 45 ml/beat/m <sup>2</sup>
Systemic Vascular Resistance (SVR)	900 – 1300 dynes-sec/cm <sup>5</sup>
SVR Index (SVRI)	1900 – 2400 dyne-sec/cm <sup>5</sup>
Pulmonary Vascular Resistance (PVR)	40 – 150 dyne-sec/cm <sup>5</sup>
PVR Index (PVRI)	120 – 200 dynes-sec.cm <sup>5</sup>

Figure 3 Measured and derived values obtained with PAC

### Cardiac output measurement

The indicator dilution method proposed by Stewart in 1890 for determining cardiac output, and later refined by Hamilton, relies on the upstream injection of a known amount of a substance and the measurement of the concentration of the substance over time by a downstream detector, giving an indicator dilution curve (shown alongside).

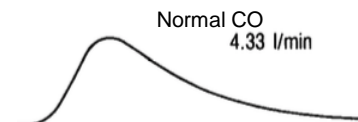


This was historically done using indocyanine green as the indicator, and cardiac output can be calculated using the Stewart-Hamilton equation:

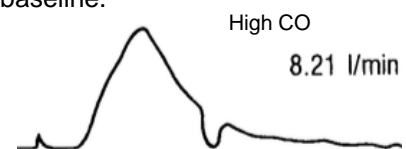
$$CO = \frac{I \times 60}{c_m \times t} \times \frac{1}{k}$$

where CO = cardiac output in l/min, I = amount of dye injected (mg), 60 = 60 sec /min,  $c_m$  = mean indicator concentration (mg/l), t = total curve duration (s) and K = calibration factor.

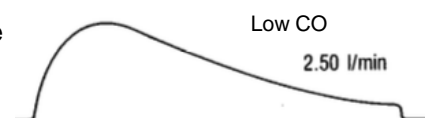
Thermodilution uses the same indicator dilution principles, using temperature change as the indicator, and has become the standard of practice for practical, bedside cardiac output measurement. Cold or room temperature solution is injected rapidly and the temperature change measured at the thermistor bead in the distal PAC. The change in temperature is negative, but is represented upright on a time-temperature curve by convention, where the area under the curve is inversely proportional to the cardiac output. A normal curve shows a sharp upstroke from rapid injection of cold fluid bolus, followed by a smooth curve returning to baseline.



With increased cardiac output, increased blood flow produces a steeper and shorter thermodilution curve.



Low cardiac output states will have a slurred curve with delayed peak, and slower return to baseline. This curve is exaggerated in tricuspid regurgitation with recirculation of injectate.



The simplified and modified Stewart-Hamilton equation becomes:

$$CO = \frac{V \times (T_b - T_i) K_1 \times K_2}{A}$$

where CO = cardiac output in l/min, V = injected volume,  $T_b$  = blood temperature,  $T_i$  = injectate temperature, K1 and K2 are corrections for specific heat and gravity of injectate and blood, and A = area under the curve (change in blood temperature as a function of time).

In order to ensure consistency and accuracy, injectate should be bolused rapidly at the end of expiration and averaged over a minimum of three measurements. Very cold injectate (0-4°C) is more accurate (better signal to noise ratio), but may cause bradycardia and a decrease in cardiac output, making room temperature injectate safer.

### Mixed venous oxygen saturation

Mixed venous oxygen saturation ( $S_{v}O_2$ ) measurement is performed on a sample from the distal lumen of the PAC and is distinct from central venous oxygen saturation ( $S_{cv}O_2$ ), which is usually from blood sampled in the superior vena cava (SVC).  $S_{v}O_2$  gives an indication of oxygen delivery and extraction, but can be high (sepsis, hepatic failure, wedged PAC, high  $FiO_2$ , decreased oxygen demand) or low (multiorgan failure, anaemia, increased oxygen demand, cardiac arrest) in varied clinical contexts, making routine monitoring of limited clinical use.

### References

1. Kumar A, Anel R, Bunnell E, et al. Pulmonary artery occlusion pressure and central venous pressure fail to predict ventricular filling volume, cardiac performance, or the response to volume infusion in normal subjects *Crit Care Med* 2004;**32**(3):691-699
2. Marik P, Cavallazzi R. Does the Central Venous Pressure Predict Fluid Responsiveness? An Updated Meta-Analysis and a Plea for Some Common Sense *Crit Care Med* 2013;**41**(7): 1774-81
3. Swan H, Ganz W, Forrester J, et al. Catheterization of the Heart in Man with Use of a Flow-Directed Balloon-Tipped Catheter *N Engl J Med* 1970; **283**:447-451
4. Sandham JD, Hull RD, Brant RF, et al. A randomized, controlled trial of the use of pulmonary-artery catheters in high-risk surgical patients. *N Engl J Med* 2003; **348**: 5–14
5. Harvey S, Harrison DA, Singer M, et al. Assessment of the clinical effectiveness of pulmonary artery catheters in management of patients in intensive care (PAC-Man): a randomised controlled trial. *Lancet* 2005; **366**: 472–7
6. Easby D, Dalrymple P. Monitoring arterial, central venous and pulmonary capillary wedge pressure. *Anaesthesia and Intensive Care Medicine* 2009; **10**(1):38-44