Pulmonary Artery Catheter Education Package

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Created April 2020

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INTRODUCTION

The pulmonary artery catheter (PAC) education package will provide you with a basic introduction to cardiac and pulmonary physiology. Building on this understanding the education package then covers the haemodynamic parameters measured and derived by the pulmonary artery catheter and how the catheter may be used in daily practice.

The pulmonary artery catheter (also known as the Swann-Ganz catheter in honour of its inventors Jeremy Swan and William Ganz) was developed in 1970. The pulmonary artery catheter is a balloon tipped multi-lumen catheter that is floated into the pulmonary artery to measure haemodynamic variables alongside calculated haemodynamic variables.

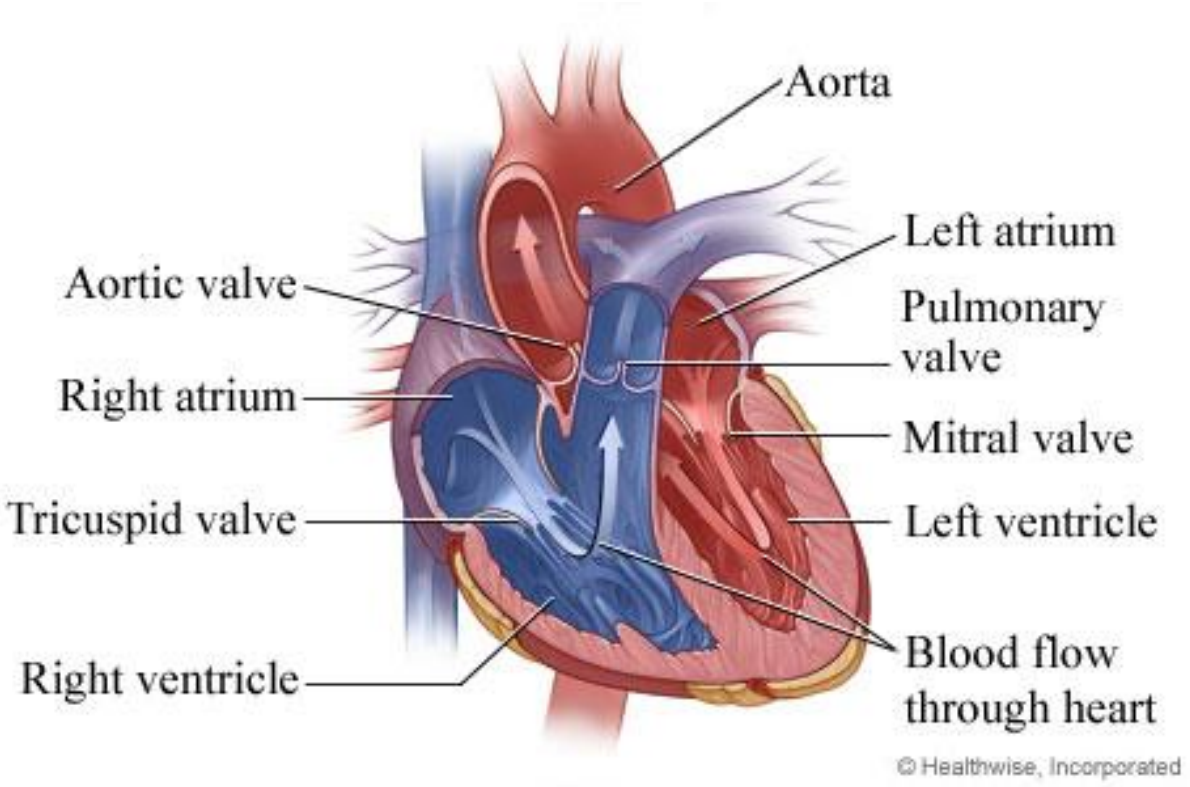
The PAC fell out of favour following the publication of the PAC-MAN trial in 2005, which reported no benefit to patients, with risk of harm, particularly in intensive care units not familiar with PACs. The study was not powered for subgroup analysis of patients in whom the PAC may benefit, for example, acute right heart failure and pulmonary hypertension. The purpose of this package is to familiarise staff with the underlying anatomy, physiology, use, and troubleshooting of PACs.

CARDIAC ANATOMY

The heart can be described as having two separate pumps. The right ventricle pumps blood into the pulmonary circulation, which is a low pressure, high capacitance system whereas the left ventricle ejects blood against a high pressure, low capacitance system.

Right heart
The right heart consists of the right atrium, tricuspid valve, right ventricle, and pulmonary valve. The right ventricle receives deoxygenated blood from the systemic circulation and pumps this blood to the lungs via the pulmonary arteries.

Left heart
The left heart consists of the left atrium, mitral valve, left ventricle, and aortic valve. The left ventricle receives oxygenated blood from the pulmonary veins and pumps this blood into the systemic circulation via the aorta.



CARDIOVASCULAR PHYSIOLOGY

Cardiac output

Cardiac output (CO) is defined as the volume of blood ejected from the ventricle per minute (litres/minute). The cardiac output from the right ventricle is the same as the cardiac output from the left ventricle.

This can then be divided by the patient's body surface area to create the cardiac index (CI).

Determinants of cardiac output

The determinants of CO are heart rate (HR) and stroke volume (SV)

$$CO = HR \times SV$$

Heart rate

Increasing HR by virtue of the equation will increase CO but in diseased hearts or at significantly elevated heart rate, cardiac output may decrease. An elevated heart rates increases myocardial oxygen consumption, reduces coronary blood flow (particularly in the left heart where coronary blood flow occurs predominantly in diastole), and reduces ventricular diastolic filling time thus resulting in a reduced blood volume to eject during systole.

Stroke volume

Stroke volume is the amount of blood ejected during systole

SV is the difference between the blood volume left at the end of diastole and the blood volume left at the end of systole.

Normal stroke volume = 60-100mls/beat

$$SV = EDV – ESV$$

Ejection fraction (EF): Stroke volume is expressed as a percentage of end-diastolic volume. Normal EF at rest = 55-70%

$$EF = SV / EDV \times 100$$

Determinants of stroke volume

Altering the determinants of stroke volume will directly alter cardiac output.

The determinants of stroke volume include:

- Preload
- Afterload
- Contractility

Preload

Preload is the amount of myocardial muscle stretch at the end of diastole, which depends on the volume of blood inside the ventricle. Measuring individual myocardial stretch is not feasible at the bedside, and measuring volume is difficult. We measure pressure inside the ventricle as a substitute for the measurement of ventricular volume, because pressure and volume are related according to *compliance.* Compliance is a property of any organ or tissue that can change its volume, and any change in volume will give rise to a change in pressure inside it. An organ that can tolerate extra volume without much rise in pressure has ‘good’ or ‘high compliance’ but if the pressure rises rapidly as volume is added, this is ‘poor’ or ‘low compliance’, and we presume we have reached the limits of volume expansion. The compliance is calculated by the change in volume divided by the change in pressure.

Clinical setting
Right ventricular end diastolic pressure can be measured directly by the pulmonary artery catheter as the catheter moves through the right ventricle.

A surrogate marker for ventricular end diastolic pressure is central venous pressure (CVP). This can be measured by transducing the proximal port on the PA catheter. These are often referred to as “filling pressures”, alongside PA diastolic pressure.

Example
Increasing preload by increasing venous return (e.g. fluid bolus) will increase end-diastolic volume and therefore result in increased myocardial stretch and increased stroke volume in a normal heart. This mechanism is explained by the Frank Starling effect.

Afterload

Afterload is the wall tension that needs to be generated by the ventricle to overcome the resistance to ejection of blood. The afterload on the right and left heart are different. The right heart has significantly less afterload imposed on it because the pulmonary circulation is a low pressure, low resistance system.

Normal pulmonary pressures = 25/8mmHg

The left ventricle has a higher afterload imposed on it because the systemic circulation is a high pressure, high resistance system.

Normal aortic pressures = 120/80mmHg

Mean arterial pressure (MAP) is used as an index of afterload.
Consequently, the left ventricle has a thicker myocardium to allow it to generate a larger force of contraction

Clinical setting
The most sensitive marker for afterload is systemic vascular resistance (SVR) for the left ventricle and pulmonary vascular resistance (PVR) for the right ventricle. SVR and PVR are derived values.

SVR = (MAP – RAP) x 80 / CO

Normal value = 800 – 1200 dynes/sec/cm5

PVR = (MPA – PAW) x 80 / CO

Normal value = < 250 dynes/sec/cm5

MAP = Mean arterial pressure
RAP = Right atrial pressure
CO = Cardiac output
MPA = Mean pulmonary artery pressure

PAWP = Pulmonary artery wedge pressure

Increasing afterload on both the right and left ventricle has an inverse relationship on ventricular function.

Example
Acute onset systemic or pulmonary hypertension increases afterload on the ventricles and reduces stroke volume and therefore cardiac output.

Contractility

Contractility or ionotropy is the inherent property of the myocardial muscle fibres to shorten and contract, resulting in ejection of blood from both ventricles.
Factors that increase contractility include increased sympathetic stimulation and inotropic agents. Factors that decrease contractility include most sedative drugs and severe acidosis.

Example
Increased contractility via administration of dobutamine increases cardiac output by increasing the stroke volume (assuming that preload and afterload are constant) as well as the heart rate.

Summary

Changes in heart rate and stroke volume will alter cardiac output. Changes in preload, afterload, and contractility will alter stroke volume and thus cardiac output. Values measured and derived from the insertion of a pulmonary artery catheter assists the clinician in making an assessment about all the factors that affect cardiac performance.

Measurement of cardiac output

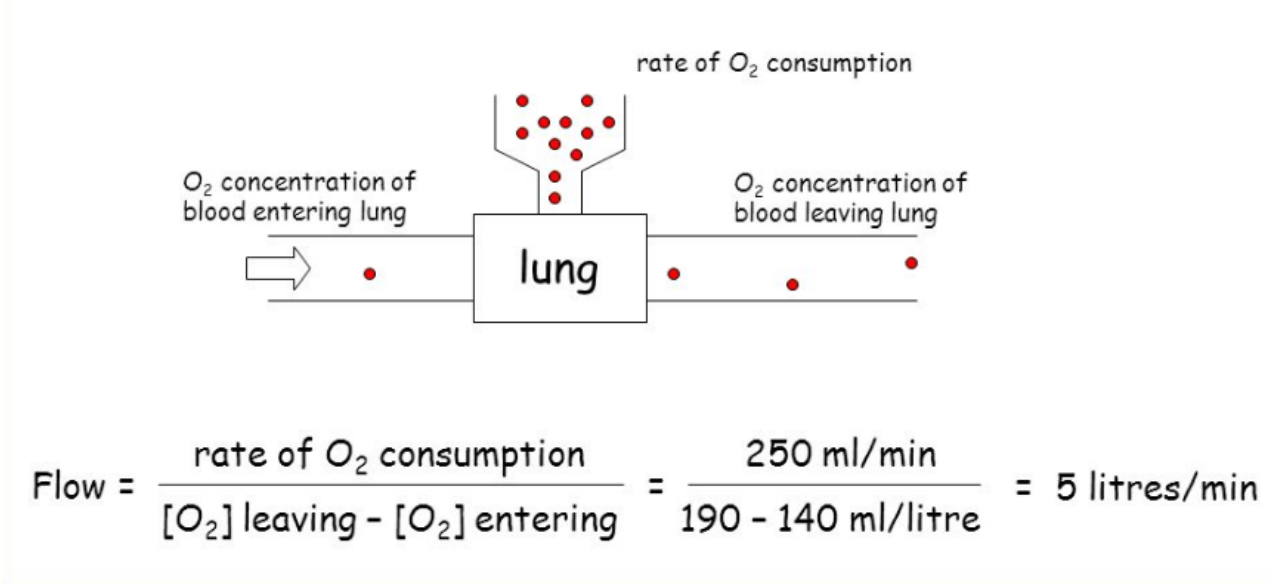
There are three main indirect methods of measuring cardiac output: The Fick method, the dye indicator dilution method, and thermodilution indicator method.

The dye indicator dilution method will not be discussed in this education package.

Fick Method

The Fick Method is the “gold standard” for measurement of cardiac output.

The concept proposes that *“the uptake or release of a substance by an organ is the product of blood flow through that organ and the difference between the arterial and venous values of the same substance.*”



The amount of oxygen entering the lung incorporates measurement of mixed venous oxygen saturation, which is measured by the pulmonary artery catheter, either by intermittent sampling or continuously.

The amount of oxygen leaving the lungs can be measured by a peripheral arterial blood gas.

Oxygen consumption is assumed to be 250mls/min

Thermodilution Indicator Method

A temperature sensing element was incorporated into the early pulmonary artery catheter that allowed for reliable and reproducible cardiac output measurement by the thermodilution method.

Calculation of cardiac output by measurement of the area under the thermodilution curve is based on the conservation of mass.

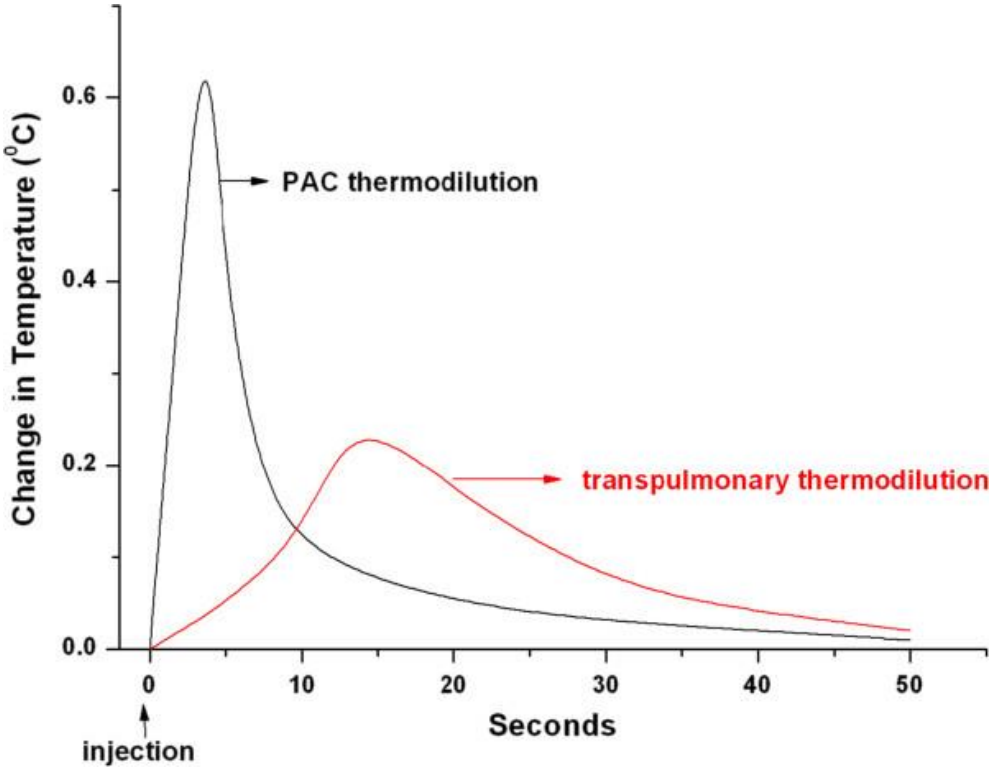
Traditional Method

1. A known amount of cold solution is injected into the right atrial lumen of the pulmonary artery catheter
2. The cold solution mixes with the warmer blood and travels through the right heart into the pulmonary artery
3. The thermistor on the pulmonary artery tip measures the change in temperature of the blood traversing the catheter tip
4. The resultant change in temperature is the plotted on a time-temperature curve

Once the PAC thermodilution curve is plotted, cardiac output can be calculated using the Stewart – Hamilton Equation (calculates the area under the curve)

$$Q = \frac{V \times (Tb - Ti) K1 \times K2}{Tb(t)dt}$$

Where:
Q = Flow
V = volume
Tb = Blood temperature
Ti = injectate temperature
K1+K2 = correction factors
Tb(t)dt = Change in temperature as a function of time



Transpulmonary thermodilution is the curve that is generated when the cold injectate travels through the pulmonary system and the thermistor is on the end of a specialised arterial catheter (hence the curve being flatter).

Measurement of cardiac output using the Edwards pulmonary artery catheter

Measurement of cardiac output using the Edwards pulmonary artery catheter utilises the same principles as the thermodilution indicator method but uses heating rather than cold injectate. The Edwards pulmonary artery catheter introduces small pulses of energy that heats the surrounding blood. The thermistor then detects the increased temperature of blood as it travels past the pulmonary artery catheter tip.

The maximum temperature of the thermal tip used to release the pulses of energy is 48 degrees.

The generation of the PAC thermodilution curve remains the same with the subsequent measurement of cardiac output using the Stewart Hamilton equation. This overcomes the disadvantages of manual injection of cold fluid, such as inter-user variability and increased infection risk.

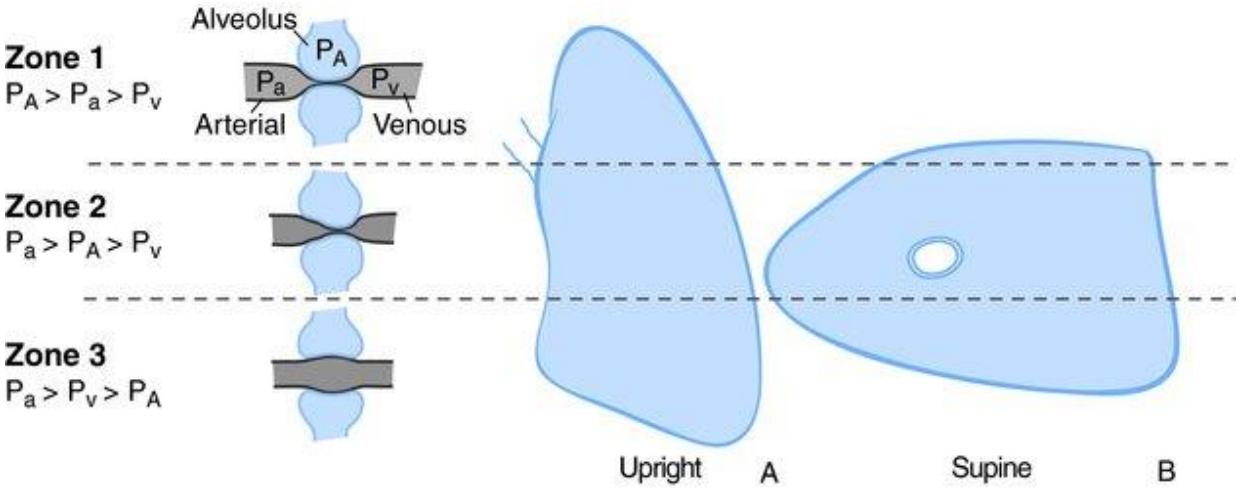
PULMONARY PHYSIOLOGY

The pulmonary artery leaving the right ventricle carries deoxygenated blood to the lungs. Gas exchange occurs at the alveolar/capillary interface. Oxygenated blood is carried via the pulmonary veins to the left atrium.

West's zones

West's zones represent different areas of the lung as described by Professor John West, a prominent respiratory physiologist. The lung can be divided into discrete zones based on the interplay of alveolar, arterial, and venous pressure. The differentiation of zones is also dependent on patient position.

In the upright position zone 1 is the apex, and zone three the base, whereas in a supine patient west zone 1 is anterior, and zone 3 more posterior.



West zone 1 is defined as a part of lung where alveolar pressure is greater than both arterial and venous pressure. West zone 1 is not found in healthy lung and more likely to develop in states where pulmonary artery pressure is too low (e.g. shock) or alveolar pressure is too high (positive pressure ventilation), or a combination of both.

West zone 2 is defined as a part of lung where arterial pressure is greater than alveolar and venous pressure. Blood flow occurs intermittently during the cardiac cycle and is dependent on the arterial – alveolar gradient. The alveolar pressure acts as a Starling resistor with flow occurring mainly in diastole

West zone 3 is defined as a part of lung where arterial pressure is greater than venous and alveolar pressure. Blood flows in both systole and diastole. West zone 3 is the location at which the pulmonary artery catheter should be placed. It represents a continuous column of blood between the pressure transducer and the left atrium.

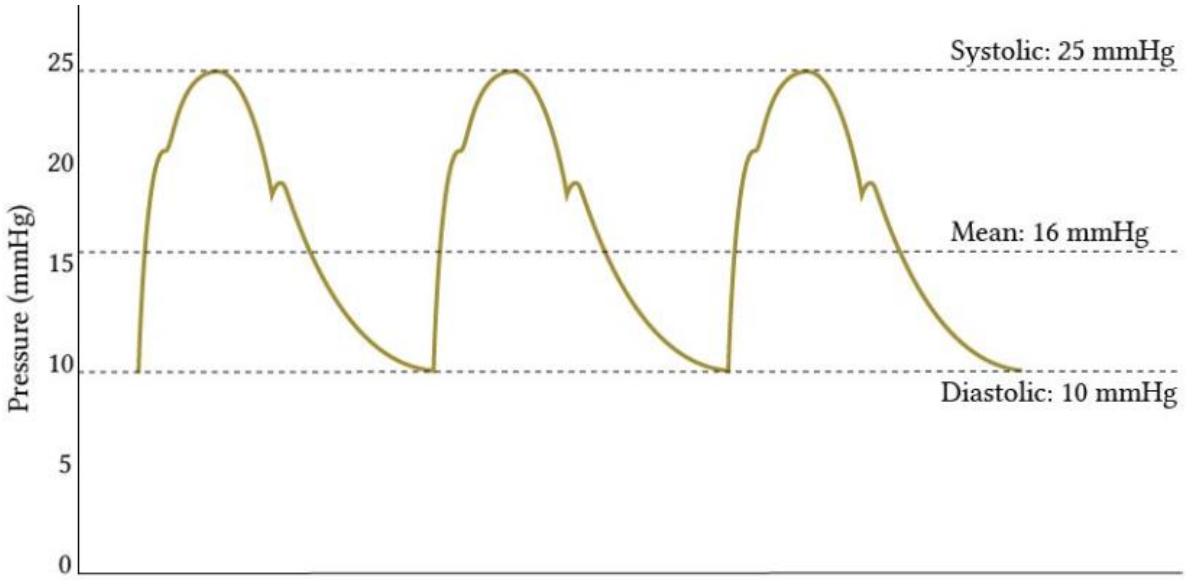
Pulmonary artery pressures

The pulmonary circulation is a low pressure, highly elastic system, with vessel walls that are much thinner and less muscular than the systemic circuit.

	Pulmonary Artery	Systemic Arteries
Systolic pressure	18 – 25mmHg	120 – 130mmHg
Diastolic pressure	8 –15mmHg	80 – 90mmHg

Pulmonary artery pressures are measured directly by the pulmonary artery catheter with a waveform displayed on the GE monitor at Nepean. These values then need to be transferred to the Edwards monitor either via slave cable or manual entry.

The following waveform represents a pulmonary arterial pressure trace



Mean pulmonary artery pressure is calculated as the area under the curve, similar to mean arterial pressure on an arterial line.

Clinical setting

Elevated pulmonary artery pressures (for example in pulmonary embolism) can increase the afterload imposed on the right heart. And due to the thin walled right ventricle, this can lead to right sided heart failure, and profound shock if left untreated.

Pulmonary capillary wedge pressure

Pulmonary artery occlusion pressure (PAOP) or pulmonary capillary wedge pressure (PCWP) is a pressure that theoretically represents the left atrial diastolic pressure.

Normal PAWP = 6 – 12mmHg

The pulmonary artery catheter is placed into a proximal pulmonary artery branch in West zone 3. When the balloon is inflated, the catheter floats further in and the balloon "wedges" in a more distal artery. The wedged catheter is now 'directly connected' to the left atrium, and the wedge pressure is theoretically the same as in the left atrium because an inflated balloon prevents blood flowing past the catheter tip.

Clinical setting

Elevated pulmonary artery occlusion pressures are indicative of elevated left atrial diastolic pressures, for example, left sided heart failure.

MIXED AND CENTRAL VENOUS OXYGEN MEASUREMENT

Mixed venous oxygen (ScVO2): blood sampled from the pulmonary artery using the distal port of the catheter

- Mixed venous oxygen saturation: the percentage of haemoglobin saturated by oxygen
- Mixed venous oxygen partial pressure: the partial pressure of oxygen dissolved in blood

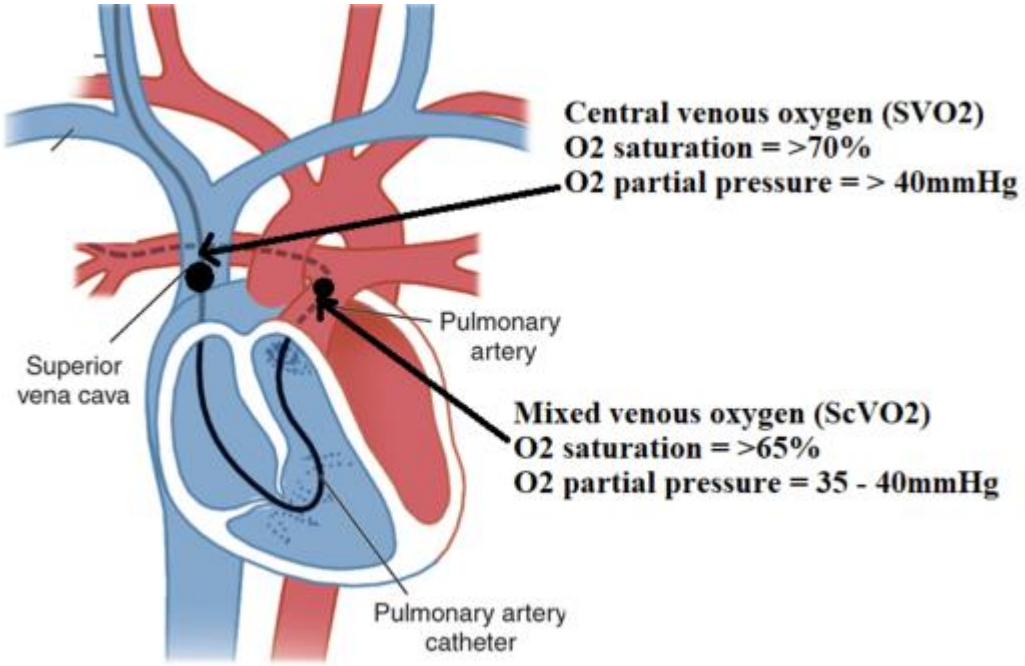
Central venous oxygen (SVO2): blood sampled from the superior vena cava using the proximal port of the catheter

- Central venous oxygen saturation: the percentage of haemoglobin saturated by oxygen
- Central venous oxygen partial pressure: the partial pressure of oxygen dissolved in blood

Anatomy of blood sampling

ScVO2 samples blood from the pulmonary artery and therefore represents true venous content because it represents mixed blood, drained from the upper body via SVC, lower body via the IVC, and the coronary circulation.

SVO2 only measures the blood from the upper portion of the body as it drains into the SVC.



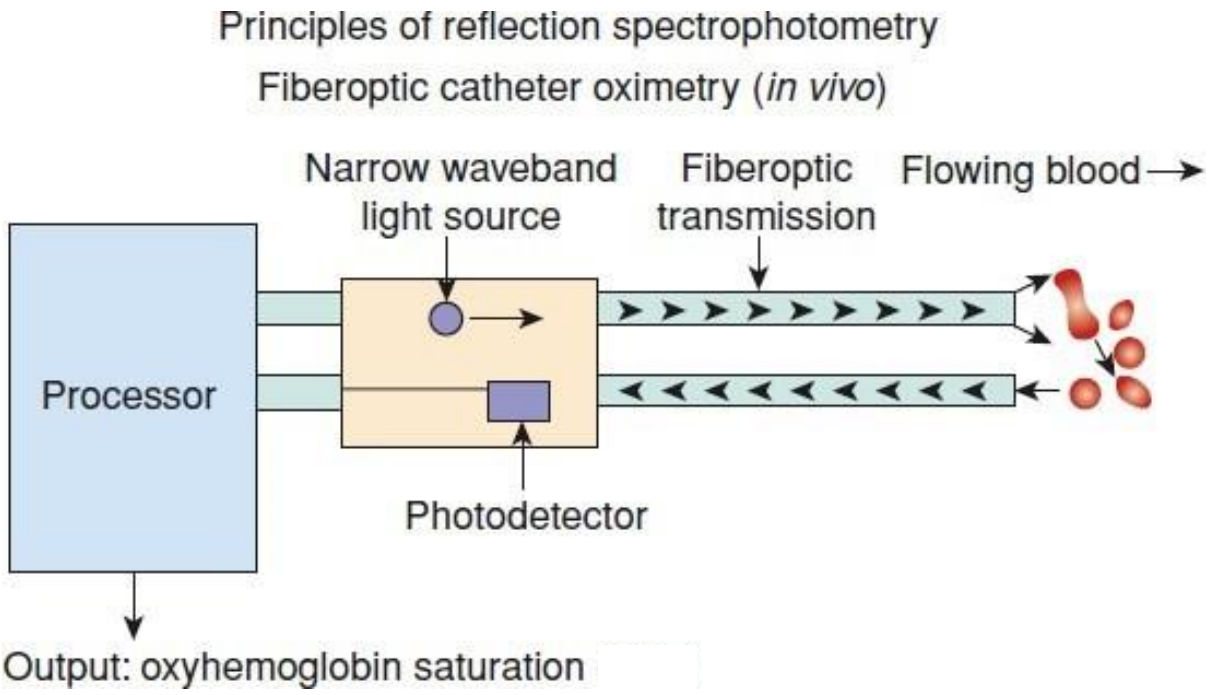
Measurement

Oxygen is both bound to haemoglobin and dissolved in plasma. Almost all the oxygen in the body is bound to haemoglobin.

Intermittent sampling of either central or mixed venous blood can be measured by a blood gas machine. Dissolved oxygen in blood is measured by a specialised electrode (Clark electrode) and oxygen bound to haemoglobin is measured by co-oximetry.

Dissolved venous oxygen = PvO2 (mmHg)
Oxygen bound to haemoglobin = SvO2 (%)

Continuous oximetry measurement by the pulmonary artery catheter measures the amount of oxygen bound to haemoglobin in venous blood. The pulmonary artery catheter emits light at different wavelengths with the reflected light measured. This represents the amount of haemoglobin saturated by oxygen and displayed as a waveform and number on the Edwards Monitor. The concept is akin to pulse oximetry used commonly in intensive care.



Physiology of ScVO2 and SVO2

Determinants of ScVO2 and SVO2

The main determinants of ScVO2 and SVO2 are oxygen consumption and oxygen delivery to the tissues. This relationship can be understood via the modification of the Fick equation.

$$ScVO2 \text{ or } SVO2 = CaO2 - VO2/CO$$

CaO2 = Arterial oxygen content (both dissolved and haemoglobin bound oxygen)
VO2 = Tissue oxygen consumption
CO = Cardiac output

Cardiac output

Changes in cardiac output lead to a directly proportional change in ScVO2 or SVO2. For example: cardiogenic shock will decrease the ScVO2 or SVO2.

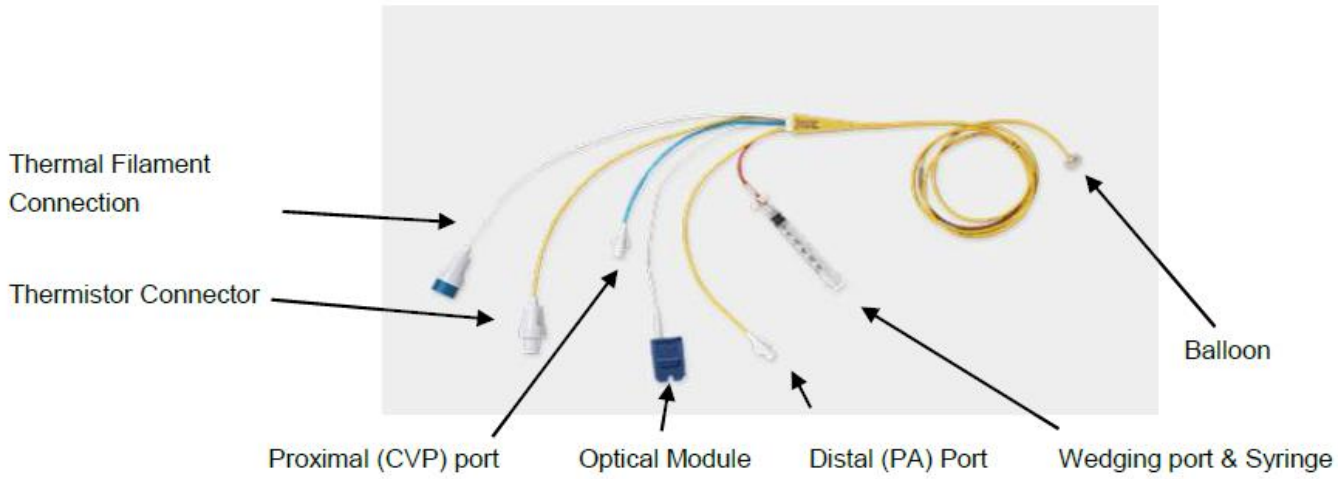
Arterial oxygen content

Changes in arterial oxygen content (both dissolved and bound to haemoglobin) havea directly proportional change in ScVO2 or SVO2. For example: anaemia results in decreased arterial oxygen content and therefore a decrease in ScVO2 or SVO2.

Oxygen consumption

Changes in tissue oxygen consumption results in an inversely proportional change in ScVO2 or SVO2. For example: increased tissue oxygen consumption (e.g. sepsis) results in a decrease in ScVO2 or SVO2.

PULMONARY ARTERY CATHETER ANATOMY



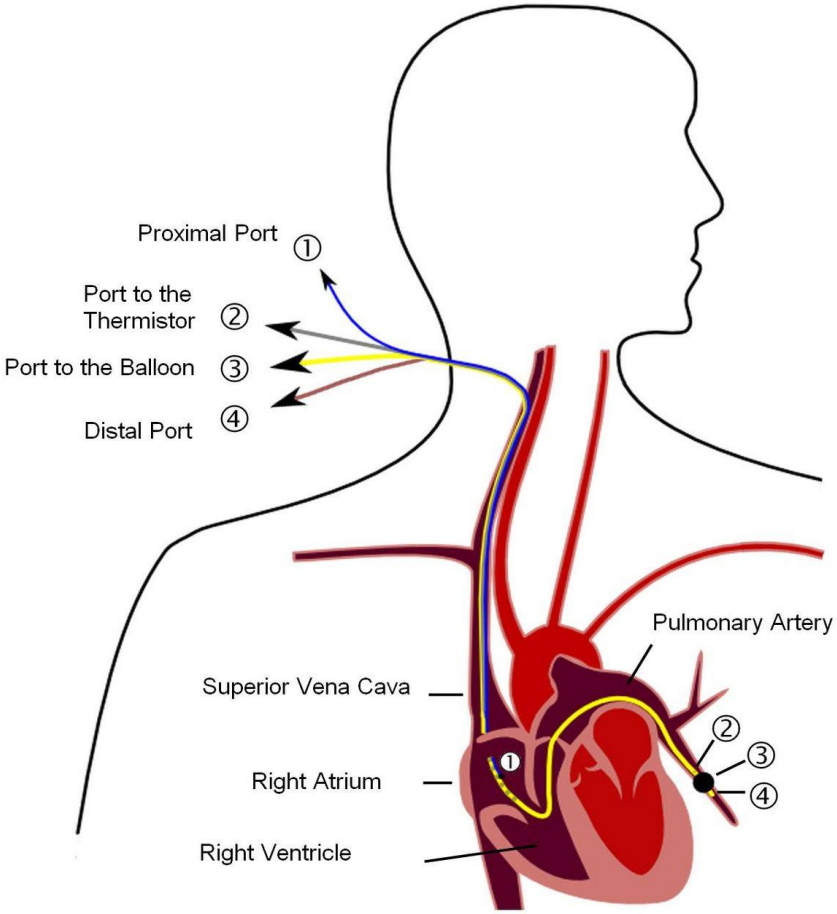
PULMONARY ARTERY CATHETERISATION

Catheterisation of the pulmonary artery requires the introduction of a pulmonary artery catheter into the superior vena cava (SVC) via an internal jugular venous sheath. Insertion of the sheath is performed via Seldinger technique under ultrasound guidance.

The PA catheter (encased in a protective sterile covering) is attached to a pressure transducer and inserted via a sterile valve through the sheath into the SVC and RA. The balloon is then inflated, and the catheter floated into the right ventricle, through the pulmonary valve into the pulmonary artery trunk, then inserted further down in a pulmonary artery branch until a wedge trace is achieved. The balloon is then deflated, the catheter withdrawn 1cm, and secured, with the length at the entry to the sheath recorded.

THE CATHETER SHOULD ONLY BE ADVANCED WITH THE BALLOON INFLATED.

ONCE WEDGE TRACE AND MEASUREMENT DONE, THE BALLOON SHOULD IMMEDIATELY BE DEFLATED. Do not inflate the balloon multiple times for one measurement



Indications and contraindications

Indications

- Measurement of pulmonary pressures
- Right ventricular failure monitoring
- Haemodynamic instability particularly in mixed shock states
- Cardiac output measurement (continuous or intermittent)
- Differentiation of cardiogenic pulmonary oedema from non-cardiogenic
- Guide to vasopressors, inotropes, fluids, and diuretic therapy
- Titration of pulmonary vasodilators in pulmonary hypertension or ARDS (e.g. nitric oxide)
- Post cardiac surgery (inserted in theatre)

Contraindications

- Coagulopathy
- Tricuspid or pulmonary valve prosthesis which can be damaged/infected
- Tricuspid or pulmonary valve vegetations that can be dislodged
- Right heart mass (e.g. tumour or thrombus)
- High risk arrhythmias (relative contraindication)

Complications

General

- Perforation of SVC
- Haemothorax, pneumothorax
- Arrhythmias
- Arterial puncture
- Haematoma at insertion site

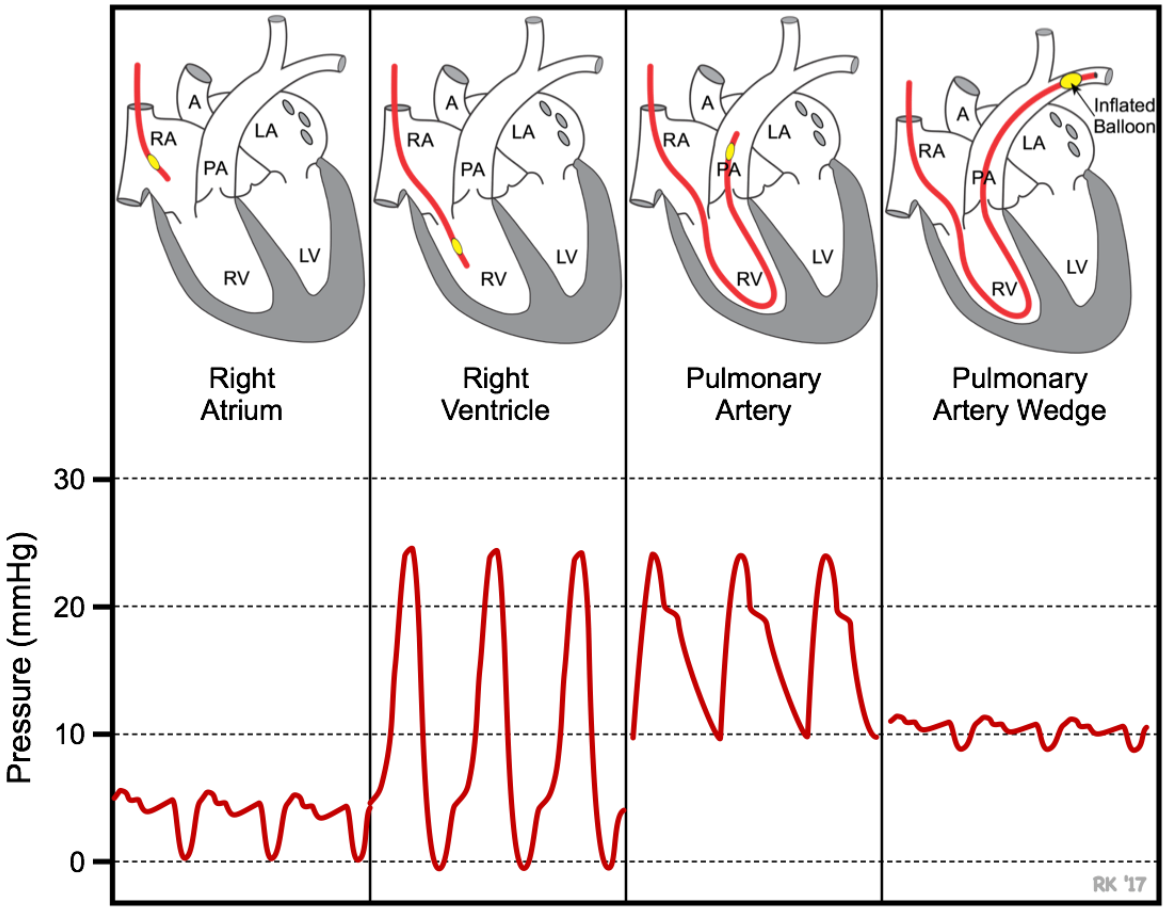
Specific

- Ventricular arrhythmia
- Thromboembolic events (the catheter is a nidus for clot formation)
- Mural thrombi in the right heart
- Air embolism from ruptured balloon
- Pulmonary infarction (if balloon inflated for too long, or catheter advanced too distally)
- Infection/endocarditis
- Right bundle branch block
    - In the setting of known LBBB this will result in complete heart block
- Coiling of catheter on itself; knotting or an inability to remove
- Valvular damage (particularly when catheter pulled back whilst balloon inflated)
- Pulmonary artery rupture

Waveform to guide catheter insertion

The pressure waveforms generated by the PA catheter helps guide insertion.

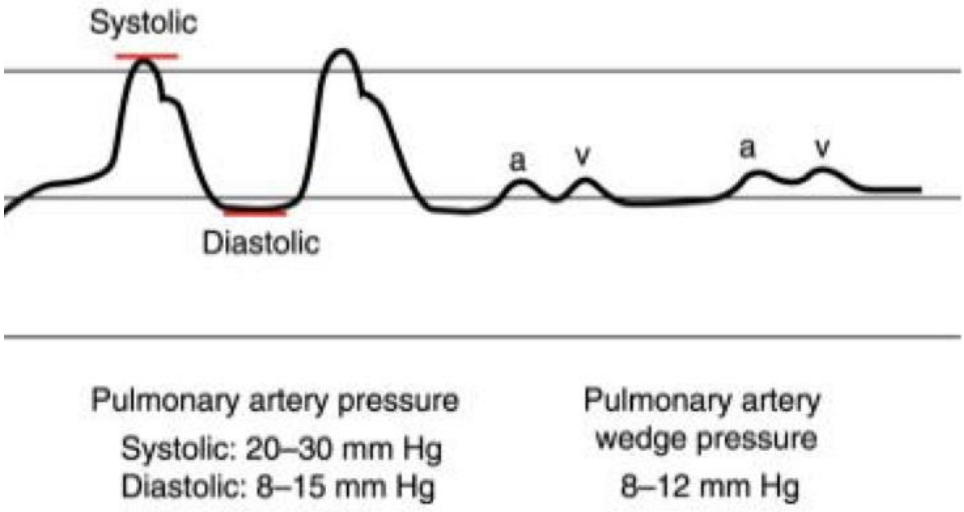
The waveform changes as the PA catheter moves from the IVC to the right ventricle, from the right ventricle to the pulmonary artery, then when wedged. The doctor inserting the PA catheter will watch the monitor to identify where the catheter is located and help determine how much further to advance the catheter.



Accidental catheter balloon tip inflation

Balloon inflation is necessary to measure the PCWP. If the intention is to not measure the PCWP and a waveform is displayed on the monitor suggesting a wedged waveform then accidental balloon inflation or catheter migration into a smaller vessel causing occlusion may have occurred. It is imperative the balloon stays deflated, unless floating the catheter into position or measuring the PCWP. If the balloon is deflated and a wedge trace is demonstrated, immediately withdraw the catheter until a PA trace is shown.

THE PAC SHOULD ONLY BE WITHDRAWN WITH A DEFLATED BALLOON

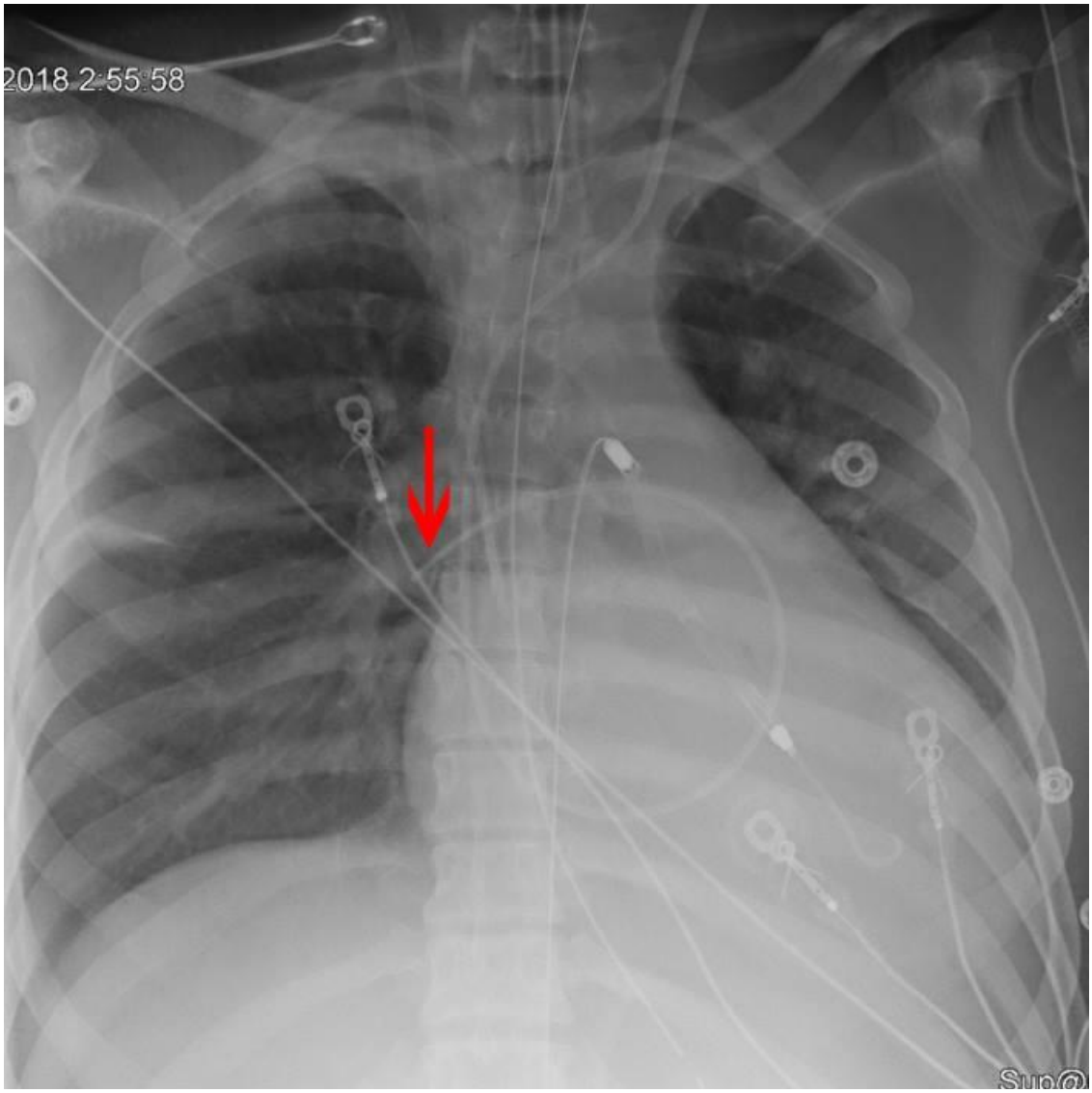


Pulmonary artery erosion and rupture may occur if the balloon is inappropriately inflated and if the inflation continues for longer than required. Consequences range between pulmonary infarction and death. The PAC specific syringe should always be left attached with the 1.5mls of air aspirated and the valve open.

Final position

Waveform
The waveform and wedging will help determine the final location.

Xray
A chest radiograph can help identify the correct location of the pulmonary artery catheter tip. It needs to be in West zone three. This represents a portion of lung with a constant column of blood between the pressure transducer and left atrium. On the x-ray it should be 2-3cm from the midline and no more than 2cm from the hilum. It also needs to be below the left atrial border.



Troubleshooting insertion problems

Advancing issues

Unable to advance catheter from SVC or RA into RV.

This could be due to tricuspid regurgitation, stenosis, or vegetation. If due to tricuspid regurgitation; inflate the balloon with 1.5mls water, lay the patient left side down and try advancing (the aim is to utilise gravity). Another method is to reduce the volume of air in the balloon to 1ml (aiming to reduce the contact of the balloon and an obstruction to passage)

Unable to advance catheter beyond the RV.

This could be due to pulmonary stenosis (rare) but more commonly due to catheter coiling in the right ventricle. Withdraw the catheter (balloon deflated) back into the SVC and try again

Wedge waveform despite balloon deflation

This occurs when the catheter tip has migrated further into the pulmonary artery and occludes the vessel despite the balloon being deflated. Withdraw the catheter until the wedge waveform disappears

Arrhythmia

Atrial and ventricular arrhythmias may occur throughout insertion of the PA catheter. They will disappear once the catheter continues to be inserted, if there is any concern withdraw or remove catheter.

If complete heart block develops then you have irritated the AV node. Withdraw the catheter immediately and get pacing equipment ready. It will be transient if only the AV node was irritated. If the heart block does not resolve, it is likely you have damaged the AV node. This will require ongoing pacing as a bridge for permanent pacemaker.

Pulmonary artery rupture

Pulmonary artery rupture carries a high mortality. The aim is tamponade the bleeding and transfer to OT or Angiography for definitive management. The following is a guideline to the immediate management-

I. Call Anaesthetics and Thoracic Surgical team
II. Lay the patient ruptured side down/PAC side down (if known)
III. Intubate with a double-lumen or single lumen tube to isolate the unaffected lung
IV. (Balloon deflated) withdraw PAC 1-2cm and then reinflate balloon (hoping to tamponade bleeding)
V. Resuscitation
VI. Repair in OT or urgent radiology intervention

Safety flags

Insertion of the pulmonary artery catheter needs to be done by an experienced medical officer. The following can help reduce the risk of complications:

- Ensure the balloon is always deflated with PAC specific syringe containing 1.5mls air and valve open
- Never withdraw the PAC with the balloon inflated – if in doubt deflate
- Know your wedge trace – immediately flag to a senior medical officer if seen
- Ensure the PAC is enclosed in the sterile covering to allow sterile re-floating
- Assess regularly for ongoing benefit from PAC and removal ASAP

HAEMODYNAMIC MONITORING

All measurements require the patient to be in the same position, ideally head up at 30 degrees. If this is not feasible then use supine position for all measurements.

Measured values

	Measurement	Clinical relevance	Values
Central venous pressure (CVP)	Attach CVP transducer cable to proximal port (blue)	Surrogate marker of right ventricular end diastolic pressure. Volume status and right ventricular function	0-6mmHg
Pulmonary artery pressure (PAP)	Attack PA transducer cable to distal port (yellow)	Calculate pulmonary artery pressures and degree of afterload on the right ventricle	25/8mmHg
Pulmonary capillary wedge pressure (PCWP)	Inflate PA catheter tip balloon with 1.5mls air	Correlates with left atrial diastolic pressure and determines left ventricular function	4-12mmHg
Core body Temperature	Thermistor on PA catheter tip measures blood temperature	Core body temperature	36-37.5 degrees
Continuous Oximetry (ScVO2)	Continuous pulsations of wavelengths used to measure oxygen bound to haemoglobin	Measures the relationship between oxygen delivery and demand. Function of cardiac output	65-75%

Derived values

	Measurement	Clinical relevance	Values
Systemic vascular resistance/ (SVR)	$\frac{(MAP - CVP) \times 80}{CO}$	Resistance in the systemic vasculature	900-1400 dynes/sec/cm^{-5}
Systemic vascular resistance index (SVRI)	$\frac{(MAP - CVP) \times 80}{CI}$	Resistance in systemic vasculature in relation to cardiac index (accounts for body surface area)	1800-2400 dynes/sec/cm^{-5}/m^2
Pulmonary vascular resistance (PVR)	$\frac{(MPAP - PAWP) \times 80}{CO}$	Resistance in pulmonary vasculature	20–120 dynes/sec/cm^{-5}
Pulmonary vascular resistance index (PVRI)	$\frac{(MPAP - PAWP) \times 80}{CI}$	Resistance in pulmonary vasculature in relation to cardiac index (accounts for body surface area)	<250 dynes · sec/cm^{-5}
Cardiac output (CO)	CO = HR x SV	The volume of blood pumped out by the heart per minute	4-8L/min
Cardiac Index (CI)	CO/BSA (body surface area)	Cardiac output in relation to body surface area	2.5-4.5 L/min/m^2
Stroke volume (SV)	CO / HR x 1000	Volume of blood ejected from the ventricle per beat	60-80mls/beat
Stroke volume index (SVI)	SV/BSA (body surface area)	Stroke volume in relation to body surface area	33 – 47 ml/beat/m2

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