Haemodynamic monitoring and management
Skills and techniques
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LEARNING OBJECTIVES

After studying this module on Haemodynamic monitoring and management, you should be able to:

1. Determine the appropriate haemodynamic monitoring for diagnosis and assessment of tissue hypoperfusion in the clinical context.

2. Describe the correct set-up of specific haemodynamic monitors and the treatments likely to be indicated by the findings.

3. Discuss the complications and limitations of haemodynamic monitors.

4. Interpret advanced haemodynamic data appropriately for diagnosis and therapy in the major types of circulatory dysfunction.

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INTRODUCTION

Haemodynamic instability is common in critically ill patients. When associated with signs of inadequate organ or tissue perfusion, whatever the cause, it may present as shock; a constellation of symptoms, signs and laboratory abnormalities that are a manifestation of tissue hypoperfusion.

Patients who survive the initial phase of shock may then develop the multiple organ dysfunction syndrome (MODS), which is a major cause of late death in the intensive care unit (ICU). Although the pathophysiology of MODS is multifactorial and not always precisely defined, haemodynamic instability, reduced organ perfusion and alterations in tissue microcirculation resulting in tissue hypoxia play key roles in the onset and maintenance of the syndrome.

Haemodynamic monitoring is necessary for assessing global and regional tissue perfusion. Timely and adequate correction of instability and tissue hypoperfusion is essential to prevent progression to MODS. Intensive care practice is characterised by a very close temporal relationship between monitoring, decision-making and treatment. Appropriate and early application of diagnostic information from haemodynamic monitoring has been shown to reduce mortality in septic shock.

1/ HOW DO I CHOOSE THE APPROPRIATE HAEMODYNAMIC MONITORING?

At the bedside, haemodynamic stability and tissue perfusion are monitored by a combination of clinical examination, monitoring devices and laboratory results. The data obtained are used to direct a clinical management plan. The focus is patient not technology centred. In practice, the monitoring devices are employed in a series of increasingly invasive and complex steps based on clinical examination and the patient’s response to treatment.

NOTE Haemodynamic monitoring per se has no favourable impact on outcome. Only the interventions based on haemodynamic data will impact outcome.

At the bedside, haemodynamic monitoring can be approached in a series of steps aimed at assessing global and regional perfusion:

Initial steps
1. Clinical assessment
2. Basic monitoring and assessment of global perfusion
3. Preload monitoring and fluid responsiveness

Advanced monitoring measures
4. Cardiac output monitoring
5. Assessment of cardiac contractility
6. Assessment of tissue perfusion.

Step 1: Clinical assessment

A clinical examination is the fastest and least invasive haemodynamic monitor available. Thirst, cold extremities, poor peripheral pulses and impaired capillary refill are useful immediate indices of hypoperfusion. A patient with inadequate global perfusion often presents with one or several of these features: tachypnoea, tachycardia, confusion, altered skin perfusion and oliguria. An awake, adequately talking patient is the best indicator of adequate cerebral perfusion. A patient complaining of ischaemic chest pain is indicating an imbalance between myocardial oxygen supply and demand. Occasionally bradycardia (heart rate <40 bpm) may be an underlying cause of low cardiac output. Oliguria in a patient with previously normal renal function and urine output is an important warning that renal perfusion is inadequate.

Particular attention should be made to detecting skin mottling. It has been shown to independently predict mortality in septic shock. Mottling usually begins at the knees, and can be quantified according to a mottling score (scored 0-5, with a higher score correlating with increased mortality). High doses of vasopressors can make skin mottling more severe and lead to purpuric changes (see images).
MOTTLING SCORE

Score 0 = no mottling
Score 1 = small area of mottling, localised to centre of knee
Score 2 = modest mottling area that does not extend beyond superior border of kneecap
Score 3 = mild mottling area that does not extend beyond the mid-thigh
Score 4 = severe mottling area, not going beyond the groin fold
Score 5 = extremely severe mottling area, extending beyond groin fold

For more information, see the PACT modules on Clinical examination and Oliguria and anuria (AKI Part I) and the following references.


Examine the next ten patients admitted to the intensive care unit and evaluate for evidence of tissue hypoperfusion. Discuss your findings with the ICU consultant or colleague.
Step 2: Basic monitoring and assessment of global perfusion

All critically ill patients should have electrocardiographic (ECG), arterial blood pressure (AP) and pulse oximetry (SpO2) monitoring. Baseline serum lactate measurements and biochemical variables should be measured.

ECG monitoring

Heart rate is an important determinant of cardiac output. Tachyarrhythmias are the commonest finding in hypoperfusion states. A 12-lead ECG performed on admission to the ICU confirms cardiac rhythm and provides baseline information on ST segments and T waves. Continuous monitoring of ST segments and the related alterations allows early recognition of myocardial ischaemia.

In patients with temporary cardiac pacing, check the underlying cardiac rhythm.

A 60-year-old female was paced via temporary epicardial pacing wires post aortic valve replacement. When pacing output was suppressed, underlying asystole was revealed and the arterial pressure trace disappeared. This patient was monitored in the High Dependency Unit until spontaneous rhythm resumed.

Blood pressure monitoring

Measuring arterial blood pressure (AP) is a cornerstone of haemodynamic assessment. The definition of low AP is patient specific and interpreted in the context of the patient’s usual AP. Mean arterial blood pressure (MAP) is an approximation of organ perfusion pressure. When stroke volume falls, MAP can initially be maintained by increasing heart rate or peripheral vasomotor tone.
Elevated AP, especially if acute, is associated with increased vascular resistance and may be associated with tissue malperfusion e.g. hypertensive encephalopathy or acute renal failure. For more information, see the PACT modules on Hypotension and Hypertension.

Arterial blood pressure may be maintained by increasing heart rate to improve cardiac output despite severe hypovolaemia, especially in younger patients.

Blood pressure may be measured non-invasively with a cuff placed around a limb and attached to a sphygmomanometer or an oscillometric device, or invasively using an indwelling catheter in an artery. Refer to Task 2.

Q. List the indications and relative indications for invasive blood pressure monitoring.

A. Indications for invasive arterial pressure monitoring:
   - Unstable blood pressure or anticipation of unstable blood pressure
   - Severe hypotension
   - Use of rapidly acting vasoactive drugs; vasodilators, vasopressors, inotropes
   - Frequent sampling of arterial blood.

Relative indications for invasive blood pressure monitoring:
   - Severe hypertension
   - Presence of an intra-aortic balloon pump
   - Patients with unreliable, or difficult to obtain, non-invasive BP.

Q. List the contraindications to invasive blood pressure monitoring.

A. Relative contraindications to invasive arterial pressure monitoring:
   - Anticipation of thrombolytic therapy
   - Severe peripheral vascular disease preventing catheter insertion
   - Vascular anomalies - AV fistula, local aneurysm, local haematoma, Raynaud’s disease
   - Lack of collateral blood flow distally (e.g. radial artery previously used for coronary artery bypass surgery).

Invasive monitoring allows beat-to-beat determination of AP. Here is an example of the variability in AP and stroke volume that occurs in atrial fibrillation.

Tissue hypoperfusion may exist in the presence of reduced, normal or elevated blood pressure.

Simultaneous recording of ECG and invasive AP trace may reveal important information about stroke volume.
ANECDOYE - A 70-year-old male presented with an exacerbation of COPD. Non-invasive AP measured in the right arm was 70/40 mmHg. Invasively-measured AP (same side) recorded similar pressure. A central venous catheter was inserted and noradrenaline (norepinephrine) infusion commenced. A nursing shift change occurred and non-invasive AP was measured from the left arm; recorded at 160/80 mmHg. The patient was weaned off the noradrenaline infusion. He had right subclavian artery stenosis secondary to peripheral vascular disease. Routinely measure AP in both arms on admission to the ICU, especially if there is discordance between clinical assessment and AP. If there is a difference consider peripheral vascular disease, aortic dissection or congenital heart disease.

Spo2 monitoring

Continuous SpO2 monitoring enables almost immediate detection of even a small reduction in arterial oxygen saturation, which is an integral part of oxygen delivery. However, based on the sigmoid shape of the dissociation curve there is a time delay of the detection of acute oxygenation failure. Taking into account the shape of the O2 dissociation curve, SpO2 should be maintained >92% in most critically ill patients. See the PACT module on Respiratory Assessment and Monitoring for additional information.

Serum lactate

The normal serum lactate level in resting humans is approximately 1 mmol/L (0.7-2.0). The value is the same whether measured in venous or arterial blood (in the absence of a tourniquet). Elevated serum lactate levels may represent poor tissue perfusion. The association of increased lactate levels with circulatory failure, anaerobic metabolism and the presence of tissue hypoxia has led to its utility as a monitor of tissue perfusion in critically ill patients.
Increased serum lactate levels at admission to ICU and a failure to normalise levels during treatment have been associated with increased morbidity and mortality.

Factors that may contribute to hyperlactataemia:
- Increased production of lactate: tissue hypoxia
- Increased aerobic glycolysis
- Inhibition of pyruvate dehydrogenase (in sepsis)
- Methanol/ethylene glycol/propofol toxicity
- Thiamine deficiency
- Decreased clearance of lactate: liver dysfunction or failure, cardiopulmonary bypass (minor reduction in clearance)
- Exogenous sources of lactate:
  - Lactate buffered solutions used in continuous veno-venous haemodiafiltration (CVVHDF)
  - Medications (metformin, nucleosidic reverse transcriptase inhibitors, long-term linezolid use, intravenous lorazepam, valproic acid
  - Haematologic malignancies.

A 45-year-old male with an acute asthmatic attack had bilateral wheeze, a peak expiratory flow (PEFR) of 150 L/min and PaCO₂ 4.0 kPa (32 mmHg), serum lactate 1.0 mmol/L. Nebulised salbutamol/iprapotropium half hourly and hydrocortisone 200 mg i.v. six hourly were given. Concern about the PEFR led to i.v. salbutamol (15 mg/kg/min) treatment. Two hours later the patient looked comfortable, had mild expiratory wheeze and PEFR measured 150 L/min. The PaCO₂ was 4.1 kPa (33 mmHg) and serum lactate 7 mmol/L. The patient was weaned off the i.v. salbutamol and within six hours serum lactate normalised. The PEFR meter was later found to be faulty. Beta2-agonists e.g. salbutamol (or adrenaline) stimulate aerobic glycolysis producing increased pyruvate which may be metabolised to lactate.

In this anecdote increased lactate level was not related to tissue hypoxia.

Bakker J. Lactate: may I have your votes please? Intensive Care Med 2001; 27(1): 6-11. PMID 11280675
An initial assessment of the circulation is completed with the use of the described monitoring tools. If tissue malperfusion is suspected, measure haemoglobin and oxygen (PaO₂) levels and treat if necessary.

**Q. Describe how oxygen is delivered to tissues.**

A. Oxygen delivery depends on blood flow (systemically regarded as cardiac output) and arterial oxygen content.

Oxygen delivery = cardiac output x arterial oxygen content.

**Q. If Hb is haemoglobin concentration and 1.39 is the volume of oxygen (mL) that combines with 1 gram of haemoglobin and SaO₂ is the percentage of Hb in arterial blood saturated with O₂ (normally 97% ± 2%), describe the oxygen content equation.**

A. Arterial oxygen content = (Hb x 1.39 x SaO₂) + (0.003 x PaO₂) per 100 mLs of blood.

Arterial O₂ content consists mainly of O₂ combined with Hb. A very small additional amount of O₂ is carried independently of Hb in physical solution. This is of the order 0.003 times the arterial oxygen tension (PaO₂); normally 95 ± 5 mmHg (12.7 ± 0.7 kPa).

**NOTE** No physical sign or haemodynamic value is absolutely specific for circulatory shock. The diagnosis should not be ruled out because a single finding, such as hypotension or lactic acidosis, is not present.

**WARNING** If hypotension or hypoperfusion is present, commence empiric therapy (e.g. i.v. fluid administration) while instituting more advanced monitoring.

**Step 3: Preload and fluid responsiveness**

In the presence of hypotension, an important step is the assessment of preload and fluid responsiveness.

Preload is defined as end-diastolic myocardial stretch (wall tension) and is often estimated at the bedside by a single/static measurement e.g. central venous pressure, CVP. More recently, assessment of fluid responsiveness (e.g. pulse pressure variation,
PPV, systolic pressure variation, SPV) has been utilised in the care of critically ill patients.

Clinically, preload may be separated into right ventricular (RV) and left ventricular (LV) preload. Jugular venous pressure (JVP) and CVP are used as surrogate estimates of RV preload. Pulmonary artery occlusion pressure (obtained using pulmonary artery catheter, see below) is used as a surrogate estimate of LV preload.

Dynamic measures such as SPV are more accurate than static measurements for assessing fluid responsiveness in mechanically ventilated patients. In simple terms, assessing fluid responsiveness asks the question: will the cardiac output increase with fluid administration? The principle behind dynamic measures is that swings in intrathoracic pressure, imposed by mechanical ventilation, affect venous return and as a consequence cardiac output. These swings in cardiac output are exaggerated in hypovolaemia indicating that the heart is operating on the ascending limb of the Frank-Starling (FS) curve.

**STATIC AND DYNAMIC MEASURE OF PRELOAD AND THE DEVICES USED FOR MEASUREMENT**

<table>
<thead>
<tr>
<th>PRESSURE</th>
<th>VOLUME</th>
<th>PRELOAD</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVP PAOP</td>
<td>GEDV LVEDV</td>
<td>PPV SPV SVV 'collapsibility'</td>
</tr>
<tr>
<td>CVP= Central venous pressure Measurement device: Central venous catheter</td>
<td>GEDV= Global end-diastolic volume (transpulmonary thermodilution) Measurement device: PiCCO™, VolumeView™</td>
<td>PPV= pulse pressure variation Measurement device: PiCCO™, LiDCOplus™, Mostcare™</td>
</tr>
<tr>
<td>PAOP=Pulmonary artery occlusion pressure Measurement device: Pulmonary artery catheter</td>
<td>LVEDV= Left ventricular end-diastolic volume Measurement device: Echocardiography</td>
<td>SPV= systolic pressure variation Measurement device: PiCCO™, LiDCOplus™, Mostcare™</td>
</tr>
<tr>
<td>IVC/ SVC 'collapsibility'</td>
<td>SVV= stroke volume variation Measurement device: PiCCO™, LiDCOplus™, Flotrac/Vigileo™, Mostcare™, Volume clamp method (e.g. Finapres™, Nexfin™), Oesophageal Doppler, Echo-Doppler</td>
<td>IVC= inferior vena cava</td>
</tr>
<tr>
<td>SVC= superior vena cava</td>
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</tbody>
</table>
Fluid responsiveness is frequently defined as an increase in cardiac output (≥ 15% from baseline) with a fluid challenge.

At the bedside, a rapid and easy way to assess fluid responsiveness is to give fluid, called a ‘fluid challenge’. A patient whose stroke volume increases following a fluid challenge is on the ascending limb of the Frank-Starling (FS) curve. In certain cases however, the patient may lie on the flat part of the FS curve, and administration of fluid may be harmful (e.g. poor LV function).

An alternative to a fluid challenge is to perform a ‘passive leg raise’ manoeuvre. This produces an ‘autotransfusion’ of blood from the venous compartments in the abdomen and lower limbs. It has the advantage of being easily reversible, and can be used in spontaneously breathing patients.

The patient is transferred from 45 degrees semirecumbent position to the passive leg raise (PLR) position, by using the automatic pivotal motion of the patient’s bed (see image below). For adequate autotransfusion to occur the patient should be maintained in the PLR position for at least one minute, when the haemodynamic effects should be observed.


For the next ten patients in the ICU receiving a fluid bolus, think about their position on the Frank-Starling curve. Observe their response to a fluid challenge and discuss your findings with the ICU consultant or colleague.

Static measures of preload: Central venous pressure

Central venous pressure (CVP) is considered a method of assessing right atrial pressure (RAP). It can be measured directly by placing a catheter in the superior vena cava. Traditionally, CVP has been used by intensivists to guide fluid management, but it is a poor predictor of fluid responsiveness and may not accurately reflect preload: due to the changes in venous tone, intrathoracic pressures, LV and RV compliance, and geometry that occur in critically ill patients, there is a poor relationship between the CVP and RV end-diastolic volume.

CVP is used frequently in ICU as a central line is often needed for other reasons (e.g. administration of vasopressors, parenteral nutrition). CVP is at best a general guide to preload with greater emphasis on dynamic values (monitoring trends in CVP over time) rather than single measurements. Despite this, it can provide important information about cardiac performance.
**Clinical use of CVP**

This can be approached in a stepwise manner:

*Observe morphology of trace*

The classic ‘a, c, v’ pattern may not always be obvious. CVP morphology may give a clue to an underlying pathological process.

**CVP CLASSIC TRACE**

![CVP Classic Trace Diagram]

*a wave= atrial contraction  
c wave= right ventricular contraction  
v wave= passive atrial filling*

**CVP SEVERE TRICUSPID REGURGITATION**

![CVP Severe Tricuspid Regurgitation Diagram]

Giant V wave: this occurs with severe tricuspid regurgitation, due to retrograde blood flow into the right atrium during ventricular systole.

*Assess value of CVP after zeroing*

Normal mean CVP = 0-5 mmHg in spontaneously breathing patient.

Upper normal limit CVP = 10 mmHg in mechanically ventilated patient.

CVP >15 mmHg = always pathological (e.g. volume overload, right ventricular failure, cor pulmonale, congestive cardiac failure, cardiac tamponade, tension pneumothorax).

*Observe response to fluid therapy*

A marked rise in CVP with fluid challenge indicates a failing ventricle.

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Other interventions may influence CVP value such as vasopressor dose change or altering patient position.
A 65-year-old lady who underwent aortic and mitral valve replacement developed hypotension suddenly on day five postoperatively. CVP rose markedly from 8 mmHg to 18 mmHg over a short period of time. This prompted resuscitation with IV fluid and vasoactive medications. Urgent bedside TTE (transthoracic echocardiography) revealed pericardial tamponade, prompting emergent sternotomy and surgical evacuation.

Q. List four other causes of an elevated CVP.

A. Acute heart failure, constrictive pericarditis, restrictive cardiomyopathy, tricuspid stenosis or regurgitation, pulmonary hypertension.

Q. List the routes of placement of a central venous catheter.

A. Central venous catheters can be inserted via several routes: internal jugular vein, subclavian vein, femoral vein.

Q. List the indications for, and relative contraindications to, insertion of a central venous catheter.

A. Indications for insertion of central venous catheter:

- Measurement of central venous pressure (providing catheter tip located proximal superior vena cava)
- Infusion of vasoactive drugs, hyperosmolar fluids (including parenteral nutrition), antibiotics, e.g. vancomycin
- Inability to obtain peripheral intravenous access
- Haemodialysis, plasmapheresis, transvenous pacing.

Relative contraindications to insertion of a central venous catheter:

- Severe coagulopathy or anticipation of need for thrombolysis
- Obvious infection of overlying skin
- Thrombosis of superior vena cava or subclavian vein.


ScvO₂ (Central venous oxygen saturation)

Insertion of a central venous catheter for CVP assessment also allows measurement of central venous oxygenation saturation, the oxygen saturation of blood in the superior vena cava. Alternatively, a ScvO₂ probe may be connected to a standard CVC for continuous measurement. ScvO₂ is a global indicator of tissue oxygenation and has been shown to be useful in guiding resuscitation in the early stages of septic shock.

NOTE ScvO₂ value <65% may indicate global tissue hypoperfusion in severe sepsis.

All measures of preload need to be interpreted in the context of the clinical condition: peripheral perfusion, urinary output and serum lactate. Single measurements should be taken in context of other variables and overall clinical condition.

Dynamic measures of preload: predicting fluid responsiveness

Dynamic preload measures are based on the ‘normal’ physiological effects of positive pressure ventilation on the right and left sides of the heart. During positive pressure inspiration, the increased intrathoracic pressure is associated with decreased venous return to the RV. At the same time, during inspiration, LV filling is increased due to compression of the pulmonary veins. This causes an increase in LV stroke volume. During expiration the LV stroke volume decreases due to reduced RV filling. These changes in LV stroke volume are most marked when a patient is hypovolaemic. The dynamic parameters include: pulse pressure variation (PPV), systolic pressure variation (SPV), and stroke volume variation (SVV).

Pulse pressure variation Pulse pressure is the difference between the arterial systolic and diastolic pressure. PPV refers to the difference between the maximum (PPₘₐₓ) and minimum (PPₘᵦᵣₜ) pulse pressure over a single mechanical breath. To document inspiration and expiration the respiratory waveform should be simultaneously measured with the arterial waveform.

PPV value can be calculated manually, or automatically using an appropriate monitoring device. It is calculated as follows:

\[
PPV\% = 100 \times \left\{\frac{PP_{\text{max}} - PP_{\text{min}}}{PP_{\text{max}} + PP_{\text{min}}} \right\}/2
\]
A PPV of \( \geq 13\% \) has been shown to be a specific and sensitive indicator of preload responsiveness.


**NOTE** Prerequisites for the adequate use of PPV include sinus rhythm, absence of spontaneous ventilatory effort (sedated), absence of right heart failure and a tidal volume \( \geq 8 \) mL/kg.

**Systolic pressure variation** The change in systolic pressure over one mechanical breath is termed systolic pressure variation. Changes in systolic pressure with mechanical inspiration may predict response to volume expansion, but with less sensitivity and specificity than PPV.


**Stroke volume variation** Stroke volume can be measured by arterial waveform analysis. It can also be measured using oesophageal Doppler technology and echocardiography.
SVV of ≥10% has also been shown to be a specific and sensitive predictor of fluid responsiveness.


**IVC/ SVC Collapsibility by transthoracic/transoesophageal echocardiography**
Positive pressure ventilation also produces change in both superior vena cava (SVC) and inferior vena caval (IVC) diameter. Cyclical changes in SVC and IVC diameter, termed ‘collapsibility’, during mechanical ventilation may therefore be used to predict fluid responsiveness.

The normal healthy heart is fluid responsive. The demonstration of fluid responsiveness is not an indication, by itself, to administer fluids. Fluid therapy should only be given if the patient is fluid responsive and there is evidence of hypoperfusion.


**Volumetric parameters: Extravascular lung water (EVLW)**
Transpulmonary thermodilution has enabled measurement of several new volumetric parameters, which can be obtained with the PiCCO™ and VolumeView™ devices. The relationship of these parameters is explained in the diagram below.
The most useful of these parameters is extravascular lung water (EVLW). This is an estimation of pulmonary oedema, the fluid accumulated in the interstitial and alveolar spaces. It is calculated indirectly from the thermodilution measurements of intrathoracic thermal volume (ITTV - see below) and pulmonary thermal volume (PTV - see below), by subtracting the intrathoracic blood volume from the intrathoracic thermal volume.

EVLW is indexed to ‘ideal’ body weight to produce an EVLW index (EVLWI) measurement. At the bedside, EVLWI measurements are useful in the detection of pulmonary oedema, and in guiding the intensivist with fluid management.

**Intrathoracic thermal volume (ITTV)** This is the volume of distribution of the thermal indicator, including: the heart (four cardiac chambers) and lungs (made up of intravascular volume, interstitial volume, and alveolar volume).

**Pulmonary thermal volume (PTV)** Consists of the intravascular, interstitial, and alveolar volumes in the lungs.
Global end-diastolic volume (GEDV) A volumetric measure of preload, and includes the volume in the four cardiac chambers. It is calculated by subtracting PTV from ITTV. GEDV is also indexed to ideal body surface area and weight, to produce Global end-diastolic volume index (GEDI) for use at the bedside.

Intrathoracic blood volume (ITBV) The volume of blood in the thoracic vasculature, including the four cardiac chambers and the pulmonary vessels. It is calculated by multiplying GEDV by 1.25. It is indexed to give an intrathoracic blood volume index (ITBI) measurement.

Pulmonary vascular permeability index (PVPI) This is the ratio of EVLW to pulmonary thermal volume, and reflects the permeability of the capillary-alveolar barrier. Thus PVPI is higher in ALI/ARDS (meaning that EVLW is high compared to PBV) than in hydrostatic pulmonary oedema.

Right ventricular end-diastolic volume (RVEDV) RVEDV is a volumetric measure of cardiac preload. A recently available pulmonary artery catheter, with a rapid response thermistor permits nearly continuous assessment of RVEDV, right ventricular ejection fraction and cardiac output.

**NORMAL VALUES FOR VOLUMETRIC PARAMETERS**

<table>
<thead>
<tr>
<th>Volumetric parameter</th>
<th>Normal values</th>
</tr>
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<tbody>
<tr>
<td>EVLWI</td>
<td>3.0-7.0 mL/kg</td>
</tr>
<tr>
<td>GEDI</td>
<td>600-800 mL/m²</td>
</tr>
<tr>
<td>ITBI</td>
<td>850-1000 mL/m²</td>
</tr>
<tr>
<td>PVPI</td>
<td>1-3</td>
</tr>
<tr>
<td>RVEDVI</td>
<td>60-100 mL/m²</td>
</tr>
</tbody>
</table>


Step 4: Cardiac output monitoring

Overview of available devices

Cardiac output (CO) monitoring plays an essential role in critical care. Direct measurement of CO should be considered when a patient remains hypotensive despite adequate fluid resuscitation or when there is ongoing evidence of global tissue hypoperfusion.

There are many CO monitoring devices available today. These include devices which use methodologies based on indicator dilution, thermodilution, pulse pressure analysis, Doppler principles, and also Fick principle. Patient status dictates the type of CO monitoring required.

Cardiac output monitoring devices

<table>
<thead>
<tr>
<th>Method</th>
<th>Monitoring System</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary thermodilution</td>
<td>Pulmonary artery catheter (PAC)</td>
</tr>
<tr>
<td>Transpulmonary thermodilution dilution</td>
<td>PiCCO™</td>
</tr>
<tr>
<td>Transpulmonary indicator dilution</td>
<td>LiDCO™</td>
</tr>
<tr>
<td>Arterial pressure waveform-derived</td>
<td>PiCCO™, LiDCO™, Flotrac/Vigileo™</td>
</tr>
<tr>
<td>Oesophageal Doppler</td>
<td>CardioQ™</td>
</tr>
<tr>
<td>Echocardiography (TTE and TOE)</td>
<td></td>
</tr>
<tr>
<td>Applied Fick (Partial CO₂ rebreathing)</td>
<td>NICO®</td>
</tr>
<tr>
<td>Bioimpedance</td>
<td>Lifegard®, TEBCO®, HOTMAN®, BioZ®</td>
</tr>
<tr>
<td>Bioreactance</td>
<td>NICOM®</td>
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</table>
Although not perfect, the pulmonary artery catheter (PAC, or right heart catheter, or Swan-Ganz Catheter) has long been considered the optimal form of haemodynamic monitoring. It allows for near continuous, simultaneous measurement of pulmonary artery and cardiac filling pressures, cardiac output, and $S\text{vO}_2$ (mixed venous oxygen saturation). Despite the relatively low risk of complications with the PAC (2-9%), the technique is invasive and its use has not been shown to clearly improve outcomes of critically ill patients (refer to PAC-Man study by Harvey et al, below). This has led to marked interest in other techniques to assess and monitor CO. Each of these newer techniques has its own limitations which need to be considered when interpreting bedside data. It must be remembered that the PAC (the ‘clinical standard’ of measuring CO) has an estimated precision of +/-20.

**NOTE** Changes in serial cardiac output determinations within 10% are within the range of measurement errors. A greater variation can be expected in patients with pronounced variability in heart rate (e.g. atrial fibrillation).


**Basic principles of thermodilution and indicator dilution methods**

The principles underlying these techniques are essentially the same. For indicator dilution (e.g. LiDCO™) a change in indicator concentration is measured over time.
The change in concentration of indicator over time produces an indicator dilution curve.

For thermodilution methods (e.g. pulmonary artery catheter, PiCCO™, VolumeView™) a drop in temperature is used instead of an injected indicator. A temperature-time curve is thus produced.
The temperature-time curves for the PAC and PiCCO™/VolumeView™ will look slightly different because of the different sites where the change in temperature is measured (pulmonary artery for PAC; femoral artery for PiCCO™/VolumeView™).
Sites of injection and temperature measurement: PAC versus PiCCO

From basic principles to bedside for thermodilution and indicator dilution methods

Pulmonary thermodilution (pulmonary artery catheter, PAC)

*Single measurement of CO:* the original PAC measures CO by an intermittent thermodilution technique. A bolus of saline at room temperature is injected into the right atrium via a port in the PAC and mixes with body temperature blood in the circulation. The change in temperature of blood in the pulmonary artery is measured using a thermistor at the tip of the PAC. The temperature drop over time is used to calculate CO.

*Continuous CO:* one type of PAC incorporates a thermal filament that warms blood in the superior vena cava (SVC). The change in blood temperature at the PAC tip is measured and provides a continuous measurement of CO (See temperature-time curve above). The displayed value represents an average of values over the previous 60-120 seconds, rather than a ‘beat-to-beat’ or ‘minute-to-minute’ measurement. The device also has a STAT mode that allows inspection of the thermodilution curve.

Transpulmonary thermodilution

The PiCCO™ (Pulsion Medical Systems, Munich, Germany) and VolumeView™ (Edwards Life Sciences) devices allow CO to be measured less invasively, using a central venous and a femoral arterial catheter, rather than a catheter in the pulmonary artery. Similar to the PAC, the devices measure a drop in temperature, using a thermistor in the arterial line, to measure the cardiac output which is then utilised for calibration—see below. Brachial and axillary lines are also available.

PiCCO™ and VolumeView™ also provide additional information that is used to calculate likelihood of developing pulmonary oedema, by calculating extravascular lung water (EVLW) - see above.
Single measurement of CO: Ice cold fluid is injected into the central line and the change in temperature measured downstream to calculate CO. Thus they are referred to as ‘transpulmonary’. This single measurement is used to calibrate the device and is recommended on set-up, every eight hours and in periods of haemodynamic instability or after adjustment of vasopressor infusion rates.

Continuous CO: this is derived by analysing the arterial pressure waveform (see below).


Transpulmonary indicator dilution

The LiDCO (LiDCO™, London, UK) device uses an indicator substance (lithium chloride) rather than a temperature drop to measure CO.

Single CO measurement: A small volume of lithium chloride is injected through a central or peripheral line and measured downstream using a lithium-selective electrode attached to the patient’s arterial line. This single measurement is used to calibrate the device and is recommended on set-up, every eight hours and in periods of haemodynamic instability or after adjustment of vasopressor infusion rates.

Continuous CO: this is derived by analysing the arterial pressure waveform (see below).

Continuous cardiac output measurement: arterial pressure waveform analysis

The PiCCO™ and LiDCO™ and Flotrac/Vigileo™ systems provide continuous CO measurement using the arterial pressure waveform. These systems analyse the arterial waveform and use algorithms to calculate the CO. The newer versions LiDCO™ (LiDCOrapid™) and Flotrac/Vigileo™ do not require calibration.

The main advantage of the arterial pressure trace-derived systems is that they are less invasive than the PAC. However they have weaknesses which limit their use in certain clinical situations.

The way in which the arterial pressure waveform is analysed is slightly different with each device. PiCCO™ analyses the systolic portion of the arterial waveform. LiDCO™ analyses the waveform with what is called pulse power analysis. Flotrac/Vigileo™ analyses the waveform 100 times/second over 20 seconds, capturing 2000 data points for analysis. This is then incorporated into a proprietary formula to calculate CO.
ARTERIAL WAVEFORM ANALYSIS METHODS

Arterial Waveform Analysis
Each of the technologies analyse the arterial waveform in a different way:

- Analyses area under systolic portion of curve: PICCO™
- Pulse power analysis: LiDCO™
- ‘Dynamic tone technology’: multiple measurements along waveform: FloTrac/Vigileo™

STROKE VOLUME

Volume clamp method This newer non-invasive technique uses an inflatable finger cuff. Photoelectric plethysmography in combination with a volume clamp technique (inflatable finger cuff) is used to produce a brachial arterial waveform, allowing continuous CO to be measured. Data to date on the usefulness of this technique in the critically ill is limited.

Volume clamp technique (Nexfin™) at the bedside
Echocardiography and Doppler technology to measure cardiac output

Echocardiography has become an important diagnostic and monitoring tool in critical care.

Cardiac output can be measured by 2D echocardiography and Doppler technology, using either a transthoracic (TTE) or transoesophageal (TOE) technique. TTE has the advantage of being rapid and non-invasive, but images may sometimes be limited in ventilated ICU patients. TOE provides high quality images but is more invasive than TTE.

Stroke volume is calculated using Doppler to measure the velocity time integral (VTi) of the flow signal at a given site, and 2D echo to measure the cross sectional area of the same site. These measurements of flow and diameter are usually obtained at the level of the left ventricular outflow tract (LVOT), and then used to calculate CO. Many modern machines will compute this information automatically when measurements are entered. Echo-Doppler calculation of CO is operator dependent, and continuous measurement of CO cannot be performed using this technique.
For further information on the use of TTE see the references below.

**Continuous transoesophageal echocardiography (hTEE™)** The hTEE™ (ImaCor inc, Garden City, New York, USA) is a miniaturised TOE probe which allows continuous qualitative haemodynamic assessment from a transverse plane, allowing visual assessment of cardiac performance and fluid status. It consists of a disposable probe (licenced for use up to 72 hours) which is connected to the echocardiography machine. Although smaller than a conventional TOE probe, some of the contraindications to TOE use may still apply with this device. There has been limited evaluation of this technique to date in critically ill patients.

**Oesophageal Doppler monitoring** Oesophageal Doppler (ODM) measures blood flow velocity in the descending aorta by using a Doppler transducer at the tip of a probe, which is inserted into the oesophagus via the mouth or nose.


**Newer devices to measure cardiac output**

**Applied Fick principle** This technique applies the Fick principle to CO₂ in order to obtain a cardiac output measurement in intubated, mechanically ventilated, and
sedated patients using a disposable rebreathing loop attached to the ventilator circuit. The method may only be applied accurately in a precisely defined clinical setting (controlled mechanical ventilation with no variation in settings, haemodynamic stability, minimal abnormality of gas exchange, minimal deadspace), and therefore its usefulness in the critical care setting may be limited.

**Electrical Bioimpedance and Bioreactance** Bioimpedance uses electrical current stimulation to identify thoracic or body impedance variations induced by cyclical changes in blood flow. CO is estimated continuously using skin electrodes or electrodes placed on an endotracheal tube, by analysing the signal variation with different mathematical models. The Bioreactance technique analyses the variations in the frequency of a delivered oscillating current occurring when the current traverses the thoracic cavity. Data on the reliability and impact on patient care of these devices in the critically ill are lacking.

**Step 5: Assessment of cardiac contractility**

Assessing cardiac contractility is important in establishing the aetiology of shock, and in guiding further therapy. For example, a patient in cardiogenic shock with poor LV function is likely to require inotropy with adrenaline or dobutamine infusion, whereas a septic patient with a hyperdynamic heart is more likely to benefit from a vasopressor infusion such as noradrenaline.

**Echocardiography**

Cardiac performance may be rapidly assessed at the bedside using transthoracic echocardiography (TTE). A visual assessment of LV function will often reveal any significant abnormality. Formal estimation of LV contractility can be performed by measuring ejection fraction (EF). The EF is the percentage of LV diastolic volume ejected with each heart beat (normal >55%).

\[
EF (%) = \frac{(EDV - ESV)}{EDV} \times 100
\]

**LEFT VENTRICULAR EJECTION FRACTION RANGES**

<table>
<thead>
<tr>
<th>Ejection fraction (EF)</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>≥55%</td>
</tr>
<tr>
<td>Mild impairment</td>
<td>45–54%</td>
</tr>
<tr>
<td>Moderate impairment</td>
<td>30–44%</td>
</tr>
<tr>
<td>Severe impairment</td>
<td>&lt;30%</td>
</tr>
</tbody>
</table>

The utility of echocardiography as a haemodynamic monitor is far greater than assessment of LV function. It is the test of choice in critically ill hypotensive patients to identify or exclude a ‘cardiac’ cause of shock as it is portable to the bedside, safe and can provide an immediate diagnosis.
Echocardiography should not be viewed simply in the context of cardiac output or ejection fraction. It can provide an assessment of preload and diagnose potentially reversible ventricular or valvular pathologies, cardiac tamponade, or massive pulmonary embolism.


**Arterial pressure waveform analysis to measure contractility**

Left ventricular contractility can also be estimated by analysis of the arterial waveform. It is derived from the maximum speed of the arterial pressure curve (dP/dt<sub>max</sub>) during the ejection phase.

**Step 6: Assessment of tissue perfusion**

Assessing the adequacy of tissue perfusion has traditionally focused on global parameters of perfusion such as: clinical examination, arterial blood pressure, urine output, serum lactate and base deficit measurements, central and mixed venous oxygen saturation.

In sepsis however, tissue hypoperfusion may result from a reduction in perfusion pressure due to both hypotension, and abnormal distribution of flow to the tissues. Regional flow to tissues is regulated by the ‘microcirculation’.

Microcirculatory failure during septic shock is characterised by oxygen shunting, vasoconstriction, tissue oedema, and thrombosis, resulting in impairment in flow distribution within the tissues. There is now strong evidence that failure of the microcirculation plays an important role in end-organ dysfunction, and has adverse prognostic implications in patients with septic shock.

**Assessing the microcirculation**

The microcirculation can be directly visualised using orthogonal polarisation spectral (OPS) and sidestream dark field (SDF) imaging devices. These devices use the principle that green light illuminates the depth of a tissue, and that scattered light is
absorbed by haemoglobin of red cells contained in superficial vessels. This enables the visualisation of capillaries and venules. These devices have been used in clinical research to evaluate the microcirculation but have not yet found a role in clinical practice.

**Near infra-red spectroscopy (NIRS)** uses the principle that the different chromophores present in skeletal muscle (such as oxy-haemoglobin, deoxy-haemoglobin, and myoglobin) have differing absorption properties of light, thus allowing tissue oxygen saturation (StO$_2$) to be derived. Non-invasive measurements of StO$_2$ using NIRS has been shown to be a reliable way of measuring the microcirculation in both septic and trauma patients. StO$_2$ measurements may be performed using a non-invasive probe either sublingually, at the thenar eminence, or at the knee (see study by Ait-Oufella et al below).

Abnormalities of the microcirculation initially or persisting following macro haemodynamic optimisation, as measured by OPS, SDF and NIRS, have been shown to be associated with poor prognosis in sepsis, trauma and general ICU patients, but targeting these regional measures of perfusion has not yet been shown to improve outcome. As a result these devices are not currently used in routine clinical practice. See the references below for further information on the microcirculation.


2/ HOW DO I SET UP THE CHOSEN TYPES OF HAEMODYNAMIC MONITORING?

Electrocardiography

Heart rate, most frequently obtained from an ECG tracing, is available simultaneously from pulse oximetry and intra-arterial blood pressure monitoring.

⚠️ Heart rate is calculated from the R-R interval and care should be taken to have the monitor distinguish the R wave from the T wave. Choose a lead where the QRS either is completely above or below the baseline and is not biphasic.

ECG calibration
ECG paper standardisation
Vertically
an impulse of 1 mV causes a deflection of 10 mm in height (2 large squares)
Horizontally
each mm (1 small square) represents a unit of time: 0.04 sec. One large square (5 small squares) = 0.20 secs

Monitoring lead systems

The monitoring lead system most commonly used in clinical practice is a 3-electrode bipolar system that can display leads I, II, III and a modified chest lead (e.g. V1). Lead II is commonly used for continuous ECG display.

3-LEAD ECG

ECG 3-lead Placements: RA (right arm): directly below the clavicle and near right shoulder, LA (left arm): directly below the clavicle and near the left shoulder, LL (left lower): on the left lower abdomen.
Alternatively, a 5-electrode system that can display the six limb leads (I, II, III, aVR, aVL, or aVF) and any one of the standard V1-V6 leads (depending on location of chest electrode) is used. Accurate ST-segment displacement monitoring requires multi-lead monitoring and precordial leads. For more information, see the PACT module on Arrhythmia.

5-LEAD ECG

5-lead ECG Placements: RA, LA, LL as for the 3-lead placement system. RL (right lower): on the right lower abdomen, C: on the chest, the position depends on the required lead selection.


Non-invasive monitoring of arterial blood pressure

**Palpation** This provides a qualitative measure of systolic arterial pressure.

**Auscultation** Brachial artery occluded by a cuff placed around the upper arm and inflated above systolic pressure. As the cuff is deflated, the return of pulsatile blood flow is accompanied by sounds that can be heard with a stethoscope placed over the artery.

In a 5-electrode ECG system, lead V1 location is most accurate for diagnosing left and right bundle branch block and for distinguishing ventricular tachycardia from supraventricular tachycardia with aberrant conduction.

Most automated non-invasive blood pressure monitoring systems use oscillometry.
**Oscillometric techniques** A cuff with an inflatable bladder is placed with the centre over the brachial artery or mid-thigh, or mid-calf. The oscillations of pressure in a sphygmomanometer cuff are recorded during inflation; the point of maximal oscillation corresponds to mean arterial pressure measurement. The oscillations begin above systolic and continue below diastolic; systolic and diastolic pressures are estimated from an algorithm.


**Invasive monitoring of arterial blood pressure**

Invasive arterial blood pressure monitoring is mandatory in shock states; it involves

- An intravascular catheter.
- A fluid-filled electro-mechanic monitoring system containing tubing, pressure transducer, and flush system.
- A monitor containing an amplifier to convert the small electronic signal generated by the transducer to a waveform that is displayed on a screen.

**HAEMODYNAMIC MONITORING SYSTEM**

The ideal artery for monitoring pressure has adequate collateral circulation. Radial, brachial, femoral and dorsalis pedis sites can be used.
The systolic AP increases progressively from the ascending aorta to the peripheral arteries. Thus, the systolic pressure may be 20-30 mmHg higher in the femoral artery than in the brachial or ulnar arteries.

**General principles of invasive pressure measurements**

The catheter is inserted into the vessel to be monitored and the external end connected to fluid-filled stiff connecting tubing. Pulsatile pressure signals at the catheter tip are propagated through the fluid in the tubing to the transducer. A pressure-sensitive diaphragm within the transducer is displaced each time it is struck by a fluid transmitted pressure pulsation. An electrical cable connects the transducer to the monitor. A pressurised flush system is designed to keep the catheter free from clots and provides a convenient means to flush or test the system. The flush bag is pressurised to 200–300 mmHg.

**Of particular importance:**

**Zero reference** ‘Zeroing’ a transducer defines a reference level from which all vascular pressures will be measured. By convention, pressures are measured relative to the level of the right atrium. ‘Zeroing’ involves opening the transducer stopcock to atmosphere and placing the air-reference port of the stopcock at the level of the midaxillary line 4th intercostal space, corresponding to the level of the estimated level of the right atrium. With the stopcock open at this level, the monitor displays 0. The stopcock is then closed to atmosphere and opened to the catheter for measurement of intravascular pressures devoid of either atmospheric or hydrostatic pressure influences. The transducer and air-reference stopcock must be kept at this level for subsequent accurate measurement of all pressures.

**Dynamic response of the fluid-filled monitoring system** refers to the ability of the system to accurately reproduce the patient’s haemodynamic waveform. Two features, resonant frequency and damping coefficient, determine the dynamic response of the monitoring system. The resonant frequency of the system is the frequency at which it oscillates when stimulated. The resonant or natural frequency of the system must be greater than the highest frequency of the incoming pulsatile signal, otherwise components of the waveform will be exaggerated. A resonant frequency greater than 20 Hertz (Hz) is needed to faithfully reproduce an arterial pressure having a frequency of 120 bpm, or 2 Hz. (a heart rate of 60 bpm has a frequency of 1 Hz). Nowadays most transducers and tubing sets are disposable and sold together, therefore the chance of connecting tubing which is inappropriate (in terms of resonance frequency) for measurement is unlikely.

**Damping coefficient** refers to how quickly the oscillating, fluid-filled system comes to rest. A system with a high damping coefficient will result in diminution of the arterial waveform. A system with a low damping coefficient will cause systolic and diastolic overshoot of the signal. Dynamic response testing is easily performed using the fast-flush test; briefly open and close the fast-flush system to produce a square...
wave that is followed by one or two rapid small oscillations before returning to baseline.

**FAST-FLUSH TEST**

![Graph showing FAST-FLUSH TEST](image)

**Q. What is the ‘damping coefficient’?**

A. Damping can be expressed as damping coefficient zeta. Zeta can be calculated for a system as follows: 

$$Zeta = 4\mu/r^3 \sqrt{\rho L/\pi E}.$$  

Where \(\mu\) = fluid viscosity, \(r\) = radius of tubing, \(SR\) = square root, \(\rho\) = density, \(L\) = length of tubing, \(E\) = elasticity of tubing. Changes in any of these elements will affect damping.

**Q. What factors may cause overdamped pressure tracings?**

A. In clinical practice an overdamped tracing (blunted) is usually caused by:

- Air bubbles, kinks or clot formation in the pressure tubing.
- Loose connections in the fluid-filled electronic monitoring system.
- Inadequate stiffness of the pressure tubing; soft low-compliance tubing results in a decrease in the natural frequency of the system such that it falls below the limit needed to record all the elements of the waveform.
- An underinflated pressure bag.

An overdamped waveform displays a falsely decreased systolic pressure and a false-high diastolic pressure, in addition to an absent or diminished dicrotic notch.

**Q. What factors may cause underdamped pressure tracings?**

A. An underdamped tracing (exaggerated) can be secondary to:

- Uses of soft, compliant tubing resulting in decreased natural frequency that may be exactly equal to one of the harmonics of transmitted pressure wave causing the tubing to vibrate more intensely. The result is artefact with overshoot of systolic pressure and ringing or vibration spikes that can obscure the waveform morphology.
• Excessive tubing length; longer tubing systems will have a lower natural frequency.
• Patient factors such as a hyperdynamic circulation (sepsis, aortic regurgitation) require a higher frequency response of the monitoring system.
• Hypertension and atherosclerosis also require a higher frequency response.
• Tachycardia: generates increased pressure signals per minute requiring a higher frequency response from the system. For example, if a patient’s heart rate increases from 60 bpm (1 Hz or pressure signal per second) to 180 bpm (3 Hz or pressure signal per second) a monitoring system that is capable of reproducing a minimum natural frequency of 20 Hz may be overwhelmed.

An underdamped wave displays a false-high systolic pressure overshoot, possibly a false-low diastolic pressure and a ringing artefact. The latter are multiple small spikes in the down stroke of the waveform.

Invasive pressure monitoring is subject to numerous potential pitfalls. If in doubt about the validity of an invasive arterial pressure reading, check the resonant frequency and damping coefficient. If possible, cross check the value using a non-invasive method at the same site.


Pulse pressure variation

Pulse pressure is the difference between arterial systolic and diastolic pressure. Refer to Task 1. In the example below, $PP_{\text{max}}$ and $PP_{\text{min}}$ are indicated by the bold lines. In this case the pulse pressure variation was calculated to be 25% indicating likely fluid responsiveness in a mechanically ventilated patient. See Task 1. The patient was in sinus rhythm and the CVP was 6 mmHg at the time of recording.

![Simultaneous recording of arterial blood pressure and respiration](image1)

Invasive monitoring of central venous pressure

Central venous pressure monitoring involves a catheter with the tip in the proximal superior vena cava and a fluid-filled electronic monitoring system to measure the pressure.

Site selection; two routes are available: internal jugular or subclavian vein. Femoral vein catheterisation allows measurements of the pressure in the inferior vena cava. Central venous pressure measurement via the femoral route may correlate with superior vena cava pressure measurement, provided the patient is in the supine position and intra-abdominal pressure is normal.


General principles of central venous catheterisation

- ECG monitoring during insertion of a central venous catheter is recommended as arrhythmias may occur during guide wire insertion.
- Full barrier, sterile technique (surgical gown, gloves and mask) is required for central catheter placement. Allow sufficient time (2-3 minutes) for the antiseptic to dry. A sterile drape should cover at least half the body to allow manipulation of the guide wire within a sterile field.
- Placing the patient in head down position allows central thoracic veins to distend and makes cannulation of the jugular or subclavian veins easier. It also reduces the risk of air embolism.
- A seeker or finder needle (22-25-gauge) attached to a 5 mL syringe may be used to locate the vein before a larger catheter/needle is used. Two-dimensional ultrasound also is useful and has been shown to reduce the rate of mechanical complications associated with central venous cannulation.
- A Seldinger technique is used for central venous cannulation. The vessel is cannulated and a guide wire advanced; the insertion site is enlarged with a small skin incision and a vessel dilator is advanced over the guide wire, presuming resistance is not encountered. The dilator is removed and the central catheter is advanced over the guide wire to the superior vena cava. The guide wire is removed and the catheter is connected to a fluid-filled monitoring system. Always ensure the guide wire protrudes through the distal end of the introducing needle/catheter to allow retrieval.

Volume resuscitation is not an indication for insertion of a central venous catheter as fluid can be delivered faster through a short wide-bore peripheral catheter.


Echocardiography

Echocardiography can be a life-saving tool in critically ill patients. Hypotension associated with a large pericardial effusion, severe LV dysfunction or acute RV dilatation can be quite easily recognised on two-dimensional echocardiography.

Information on echocardiography training, courses and accreditation is available from the sources below. The expert statement provides a consensus document on standards for critical care echocardiography training.

Expert Round Table on Ultrasound in ICU. International expert statement on training standards for critical care ultrasonography. Intensive Care Med 2011; 37(7): 1077-1083. PMID 21614639


http://www.esicm.org/
http://www.intensive.org/
http://www.asecho.org/
http://www.escardio.org/bodies/associations/EAE

Pulse contour analysis

**PiCCO™ plus**

See transpulmonary thermodilution technique (Task 1 and below).

**LiDCO™ plus**

**Equipment** A peripheral arterial catheter, calibration disposables (lithium sensor, lithium chloride ampoule (0.15 mmol/mL), disposable blood collection bag), flow regulator pump (battery operated), stand-alone LiDCO haemodynamic monitor.

**Calibration** involves measurement of the CO via indicator dilution technique in which 2 mL (0.3 mmol) of lithium is injected into a central or peripheral venous catheter. The arterial catheter is opened to the lithium sensor via a three-way stopcock and flow regulator pump allows the blood to pass the sensor at a determined rate. A lithium dilution curve is generated which serves as a calibration CO. Calibration should be performed once each shift and before initiating any major treatment changes.
Continuous stroke volume and cardiac output beat-to-beat stroke volume is calculated based on an algorithm which uses the calibration CO measurement and harmonic waveform analysis (Fourier transformation). CO is determined from the computed stroke volume and heart rate.


http://www.lidco.com/archives/LiDCOplus_brochure_1914.pdf (fig. 1 LiDCO injection site; fig 2 LiDCO arterial line site)

**Volume clamp method (e.g. Finapres™, Nexfin™)**

**Equipment** Inflatable finger cuff (consisting of bladder with an infrared plethysmograph) and stand-alone monitor.

**Calibration** The cuff is attached to the middle phalynx of the finger, and the system ‘zeroed’ at the level of the right atrium.

Beat-to-beat continuous blood pressure is measured by repeated cuff inflation. Continuous stroke volume and cardiac output is calculated from the systolic pressure area using a physiological three-element Windkessel model. Stroke volume variation and pulse pressure variation are also measured.

**Transpulmonary thermodilution technique**

**Equipment** A standard central venous catheter and a thermistor-tipped arterial catheter.

**Site** The arterial catheter is inserted in the femoral artery. The brachial or radial artery may be used in patients where femoral cannulation is contraindicated e.g. aorta-femoral bypass.

**Calibration** Transpulmonary thermodilution measurement requires the central injection of a cold (<8 °C) or room temperature (<24 °C) saline bolus, with a temperature sensor attached to the central venous catheter. The thermistor on the arterial catheter measures the downstream temperature change. Cardiac output is calculated in the usual way from the area under the thermodilution curve (modified Stewart-Hamilton algorithm).
Cardiac output Continuous measurement of CO by the pulse contour method is calculated using an algorithm measuring the area under the systolic part of arterial pressure curve (Wesseling’s method). See Task 1.

PICCO FEMORAL ARTERY SITE.     PICCO CENTRAL VENOUS SITE

Measuring static and dynamic volumetric parameters
Static volumetric parameters, including ITBV and GEDV, are obtained by advanced analysis of the thermodilution curve. For the calculation of volumes, mean transit time and down slope time of the thermodilution curve are important. Mean transit time is the time when half of the indicator has passed the point of detection in the artery. Down slope time is the exponential down slope time of the thermodilution curve. EVLW is calculated using the same approach. See Task 1 step 5. Dynamic parameters of preload, PPV and SVV are automatically calculated on a beat-to-beat basis using the pulse contour analysis.

Cardiac contractility Left ventricular contractility can be assessed by measurement of $dP/dt_{max}$, derived from the maximum speed of the arterial pressure curve during the ejection phase.

Pulmonary artery catheter
The PAC is passed aseptically through a sheath introducer device, which may be inserted from internal jugular, subclavian or, less frequently, femoral approaches. There are several types of pulmonary artery catheter (PAC) available grouped according to their monitoring capabilities:
- **Basic thermodilution model**: measurement of right atrial pressure (RAP), pulmonary artery pressure (PAP), pulmonary artery occlusion pressure (PAOP) with or without intermittent thermodilution CO measurement.
- **Basic thermodilution model with addition of infusion ports**: Basic thermodilution catheter with additional lumens that open into right atrium or right ventricle for continuous drug/fluid infusions.
- **Continuous cardiac output catheters**: measurement of RAP, PAP, PAOP plus a thermal filament within the catheter that provides continuous stroke volume and CO measurements.
- **Oximetry catheters**: with fibre optics allowing continuous monitoring of mixed venous oxygen saturation (SvO₂). Fibre optic bundles within the catheter transmit and receive red light near the catheter tip that allows measurement of saturated haemoglobin in the mixed venous blood.
- **Right ventricular volumetric catheters**: additional measurement of right ventricular ejection fraction (RVEF) via a fast response thermistor that can sense beat-to-beat temperature change. Derived parameters include continuous right ventricular end-diastolic volume (CEDV).

Below is a picture of a thermodilution pulmonary artery catheter, designed to measure right atrial (RAP) and pulmonary artery pressures (PAP) and provide continuous CO readings. Catheter markings occur every 10 cm (thin black line), the 50 cm mark is denoted by a thick black line. A distal port opens to a lumen running the length of the catheter and terminating at the tip. This port measures PAP and PAOP; mixed venous blood may be drawn from this port when the catheter tip lies within the pulmonary artery. The balloon inflation port opens to a lumen that terminates within the balloon located at the tip of the catheter. The balloon is inflated with 1.0-1.5 mL of air to facilitate passage through the heart and to wedge the catheter to obtain a PAOP measurement. A proximal port opens 30 cm from the distal tip and is used for RAP monitoring and infusion of fluids. Another proximal infusion port is located 26 cm from the distal tip. A protective sheath is extended along the external length of the catheter and attached to the introducer.

This catheter has a 10 cm long thermal filament that delivers pulses of heat which thus heat up the surrounding blood. A thermistor located 4 cm from the catheter tip detects blood temperature changes and correlates the data with the right ventricular thermal input to produce a thermodilution curve.
**Flotation of the pulmonary artery catheter**

As the catheter is passed from the central venous circulation through the heart, pressure waveforms characteristic of the site being traversed are recorded; the shape of the haemodynamic waveform and the pressure measurement are noted.

The natural curve of catheter may help flotation. Difficulty with flotation may occur when cardiac output is low (forward flow is limited), or with significant tricuspid valve disease (for example, severe TR may limit forward flow), severe pulmonary hypertension, or dilated right ventricle.

<table>
<thead>
<tr>
<th>Distance from insertion site in cm</th>
<th>Pressures mmHg systolic/diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Subclavian/internal jugular</strong></td>
<td><strong>Femoral</strong></td>
</tr>
<tr>
<td>Right atrium</td>
<td>10-15</td>
</tr>
<tr>
<td>Right ventricle</td>
<td>20-25</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>35-50</td>
</tr>
<tr>
<td>Pulmonary artery occlusion pressure</td>
<td>variable</td>
</tr>
</tbody>
</table>

In the diagram below, pressure waveform changes are noted as the catheter is advanced from the right atrium (RA) to the PAOP position.
In this case, the cardiac rhythm is atrial fibrillation. A RAP waveform (similar to the CVP trace) is observed at 15-20 cm from an internal jugular insertion site. The balloon is inflated with 1.0 mL of air and the catheter advanced. On entering the right ventricle (RV) there is a change in waveform morphology and an increase in peak pressure. The RV is entered at approximately 25-30 cm from an internal jugular insertion site. The RV waveform has a steep slope, a peak pressure that is 2-3 times higher than the mean RA pressure, and returns to a baseline RV end-diastolic pressure equal to the mean RAP. (Note the ventricular ectopic beat when the catheter is in RV). As the catheter crosses into the pulmonary circulation the baseline of the waveform rises and a dicrotic notch can be observed on the down slope. The pulmonary circulation is usually reached 40-55 cm from an internal jugular insertion site. When the catheter is advanced into a segment of a pulmonary artery smaller than the inflated balloon, forward flow is interrupted (‘wedge position’) and the distal lumen records the pressure originating from the left atrium; this is the pulmonary artery occlusion pressure (PAOP). The shape of the PAOP waveform changes to a low amplitude pressure waveform with a sine wave appearance similar to the RAP although slightly higher.

When the balloon is deflated, the PAP waveform should reappear promptly. If this does not occur, the catheter should be withdrawn several centimetres to assure that it does not remain in a wedge position causing interrupted pulmonary blood flow.

Never withdraw the catheter with the balloon inflated as it may damage cardiac structures.


Position of the catheter

A chest radiograph should be taken to confirm the position of the PAC (without any loops) and to rule out a pneumothorax. The catheter tip should not extend beyond
the pulmonary hilum as in the first chest X-ray (CXR 1) below. In CXR 2, the PAC was considered to be too distal and was withdrawn a few centimetres even though a PAP waveform was transduced.

Q. Explain what is meant by West’s lung zones.

A. West’s lung zones are a theoretical concept based on the fact that gravity influences blood flow within the lungs; pulmonary blood flow and vascular pressures increase progressively down the lung. Originally described by the pulmonary physiologist John B. West.

Q. Describe Zone 1 and its effect on the PAOP.

A. In a supine patient this is directly underneath the anterior sternum. In an erect patient Zone 1 corresponds to the lung apex. In this zone, alveolar pressure exceeds pulmonary artery and pulmonary venous pressure. Thus, a catheter wedged in this location would record alveolar pressure instead of reflecting left atrial pressure as the pulmonary veins would be completely collapsed.

Q. Describe Zone 2 and its effect on PAOP.

A. It lies directly underneath Zone 1. In this zone, alveolar pressure exceeds pulmonary artery diastolic pressure and pulmonary venous pressure. A catheter wedged in this position would record alveolar pressure, again because pulmonary veins would be collapsed.

Q. Describe Zone 3 and its effect on PAOP.

A. This is the most dependent portion of the lung in the supine or erect position. The pulmonary artery systolic and diastolic pressures and pulmonary venous pressures are always greater than alveolar pressures in this zone. The pulmonary vessels do not collapse and a catheter wedged in this position accurately measures PAOP.

A zoning artefact occurs whenever alveolar pressure exceeds pulmonary venous pressure and the recorded measurement reflects alveolar pressure rather than pressure in a pulmonary vein. Conditions that may lead to such an artefact include hypovolaemia, PEEP and position of the tip of the PAC in a West Zone 1 or 2. If the
PAC lies in a vessel below the level of the left atrium it is almost always in a Zone 3 area.

**Q. Describe a checklist for verifying position of PAC in Zone 3.**

A. PAD >PAOP, catheter tip location below the level of the left atrium on a portable lateral chest X-ray, A and V waves visible within trace (cardiac ripple), change in PAOP less than half the change in PEEP during a PEEP trial.

On the American Thoracic Society’s website below, you will find a Pulmonary Artery Catheter Primer [Clinical information/Critical care/Hemodynamic monitoring/Pulmonary artery catheter primer](http://www.lcs.mgh.harvard.edu/projects/pacath.html)

http://www.thoracic.org

**Mixed venous oxygen saturation (SvO₂)**

A true mixed venous sample (called SvO₂) is drawn from the tip of the pulmonary artery catheter, and includes all of the venous blood returning from the head and arms (via superior vena cava), the gut and lower extremities (via the inferior vena cava) and the coronary veins (via the coronary sinus). By the time the blood reaches the pulmonary artery, all venous blood has ‘mixed’ to reflect the average amount of oxygen remaining after all tissues in the body have removed oxygen from the haemoglobin. Mixed venous oxygen saturation (SvO₂) can help to determine whether the cardiac output and oxygen delivery is high enough to meet a patient’s needs. It can be very useful if measured before and after changes are made to cardiac medications or mechanical ventilation, particularly in unstable patients. Normal SvO₂ is 60-80%. If the SvO₂ is low (<60%) it means either that oxygen delivery is reduced (decreased Hb, hypoxia or decreased cardiac output), or that oxygen consumption is increased (shivering, hyperthermia or seizures).

**Measuring PA and PAO pressures**

The transmural pressure of pulmonary vessels is most reflective of the filling pressure and is the pressure within the pulmonary vessel minus the extravascular pressure i.e. the pleural pressure. As it is not possible to measure pressure surrounding pulmonary vessels, it is assumed that the pleural pressure is closest to zero at end-expiration. Therefore, all intrathoracic vascular pressures (RAP, PAP and PAOP) are measured at end-expiration. In the spontaneously breathing patient, these pressures are at their highest at end-expiration and the measurements should be made at this point, just before the pressures drop with spontaneous inspiration. During mechanical ventilation, these pressures will be lowest at end-expiration and, therefore, pressure measurements should be made just before the pressures rise with positive pressure inhalation. The end-expiratory point of the trace is PAOP measurement is usually chosen with the help of a software package allowing cursor manipulation of a marker line.

Although not precisely the same, PAOP and PCWP (pulmonary capillary wedge pressure) are used interchangeably. PAOP is the pressure measured from the distal
tip of the catheter with the balloon inflated and correlates with LA pressure. PCWP is obtained when the catheter is wedged with the balloon deflated and hence is closer to capillary pressure.

Accurate, reliable measurement of haemodynamic pressures relative to the respiratory and cardiac cycles requires the ability to either freeze the monitor sweep, or, preferably, to acquire a paper printout of the ECG and corresponding pressure waveform from a 2-channel paper recorder.

How to measure RAP and PAOP waveforms using the ECG

In RAP and PAOP waveforms with A and V waves that are of similar height, it is permissible to simply average the high and the low of the two waves at end-expiration. However, frequently the V wave of the RAP and, more commonly, the PAOP, is dominant and elevated. In these situations, measuring the average of both waves would provide an overestimate of the ventricular filling pressure. Measurement of the RAP and PAOP in these conditions requires measuring the average of only the A wave, which can only be done when the pressure waveform is correlated to the simultaneously obtained ECG. The A wave of the RAP can be located in the PR interval of the ECG, while the A wave of the PAOP can be found at the end of or immediately after the QRS complex.

PA systolic pressure occurs within the T wave of the ECG while the end-diastolic pressure occurs at the end of the QRS. Routinely assessing all haemodynamic waveforms with the ECG is an invaluable way to avoid the consequences of misinterpreting a PAOP waveform with a high, dominant V wave for a PAP. If the peak occurs well after the T wave, the waveform is that of a PAOP with a high V wave, and not a PA waveform.

Confirming accurate PAOP measurement

Several factors must be assessed to ensure that the PAC is correctly positioned to measure the PAOP accurately.

- Change in the waveform from pulmonary artery (with systole and diastole) to a PAOP waveform. Although distinct A and V waves may not always be identifiable, a low amplitude, oscillating baseline should be visible. A PA waveform should immediately return on balloon deflation.
- Fall in mean pressure; normally, the mean PAOP is similar to or slightly lower than the diastolic PAP (usually within 0-4 mmHg). However, in conditions in which a large V wave is present in the PAOP waveform, the displayed mean value of the PAOP may be higher than diastolic PAP.
- Blood aspirated from the distal tip of a PAC in the wedge position is highly oxygenated and resembles arterial blood. The PO₂ is slightly higher than PO₂ of the systemic arterial blood.
- The catheter should flush easily to exclude catheter obstruction.
In the ICU, identify five mechanically ventilated patients with pulmonary artery catheters in situ. Note the difference in pressure readings between end-inspiration and end-expiration. The value that is closest to transmural pressure is the end-expiratory reading.

**Effect of PEEP on measurement of PAOP**

PEEP (including autoPEEP) increases intrapleural pressure (during all stages of the respiratory cycle) causing the measured PAOP to overestimate the actual transmural or filling pressure. With normal lung and chest wall compliance, approximately 50% of the applied PEEP is transmitted to the pleural space and PAOP will rise by less than 50%.

**NOTE** If PAOP increases >50% of the amount of applied PEEP, the measurement may be in error or the catheter may be malpositioned.

The normal pulmonary vascular network is a low-resistance circuit. Different factors affect the respective pulmonary artery pressures.

**Q. Which factors have the effect of increasing pulmonary artery ‘systolic’ pressure?**

A. Any situation where there is increased pulmonary vascular resistance e.g. hypoxaemia, pre-existing chronic lung disease e.g. COPD, pulmonary embolism, ARDS, sepsis, primary pulmonary hypertension, large left-to-right shunts e.g. ASD, VSD.

**Q. Which factors have the effect of increasing pulmonary artery ‘diastolic’ pressure?**

A. All conditions where systolic PAP is elevated:
   - Hypervolaemia
   - Left heart dysfunction of any cause; LV failure, mitral stenosis/regurgitation, decreased LV compliance
   - Cardiac tamponade
   - Constrictive or restrictive pericarditis.

**Q. How do you calculate pulmonary vascular resistance (PVR)?**

A. Pulmonary vascular resistance can be calculated as:

\[
\text{mean} \left( \frac{\text{PAP} - \text{PAOP}}{\text{cardiac output}} \right)
\]
Q. Name clinical conditions where PAOP does not accurately reflect left ventricular end-diastolic pressure

A.

- Aortic regurgitation
- Any condition that produces obstruction in the pulmonary veins e.g. tumour, fibrosis, thrombosis
- Left atrial mass e.g. large myxoma, thrombus
- Mitral valve pathology; stenosis or regurgitation with elevated V wave
- Significant tachycardia (heart rate >130 beats/min)
- Increased pleural pressure (e.g. PEEP, CPAP especially with associated hypovolaemia or in patients with emphysema)
- Catheter placement in West Zone 1 or Zone 2 of the lung—see below.

Some conditions result in a discrepancy between PAOP and left ventricular end-diastolic pressure (LVEDP).

Q. When will the measured mean PAOP be lower than the true LVEDP?

A. Decreased left ventricular compliance e.g. left ventricular hypertrophy or myocardial ischaemia.

Q. When will the measured PAOP be greater than the true LVEDP?

A. Reduction of pulmonary tree; pneumonectomy, massive pulmonary embolism.


**ANECDOCTE** A mechanically ventilated patient with a PAC in situ has a central venous catheter inserted (via left subclavian vein) for renal replacement therapy. Two hours post-insertion, blood pressure falls rapidly and CVP, PAP and PAOP increase while CO decreases.

It is noticed that CVP, diastolic PAP and PAOP values are almost equal. The absence of normal breath sounds and hypertympanic percussion over the left thorax suggested the diagnosis of a tension pneumothorax. A chest tube was inserted and air drained with immediate improvement in blood pressure. Haemodynamic values must be interpreted in the context of each patient’s history and physical findings.
3/ LIMITATIONS AND COMPLICATIONS OF HAEMODYNAMIC MONITORING

Electrocardiography

Like most clinical tests the ECG yields both false positive and false negative results. It is of great importance in clinical practice to be aware of these diagnostic limitations.

Conditions not excluded by a normal or non-diagnostic ECG:
- Prior MI
- Acute MI (the more common scenario is NSTEMI)
- Severe coronary artery disease
- Significant left or right ventricular hypertrophy
- Intermittent arrhythmias (e.g. ventricular tachycardia)
- Hyperkalemia
- Acute pulmonary embolism may be masked in presence of left bundle branch block, pacemaker pattern ECG.

Any ECG findings should be correlated with clinical observation of the patient.

Q. When may ECG signs be falsely positive for LVH?
A. High voltage in the chest leads may be a normal finding, especially in young adult males with thin chest walls. Therefore, high voltage (Sv1 + Rv5/Rv6 >35 mm) is not a specific indicator of left ventricular hypertrophy and the diagnosis should not be made on this finding alone.

Q. When may ECG signs be falsely positive for myocardial ischaemia?
A. Q waves may occur as a normal variant and do not always indicate heart disease. Q waves normally occur in leads I, aVL, V4, V5 and V6. They are narrow with no notching or slurring and are the result of septal activation. With normal variance, the depth of the Q wave is <2 mm (2 small squares) in leads I and II and is rarely more than 1 mm in depth in any of the other leads.

Early repolarisation variant. Slight deviations in the ST segment (<1 mm) may be seen in healthy people. Occasionally a benign variant called early repolarisation can be seen mostly in young although occasionally in older patients. With early repolarisation the ST segments may rise up to 3 mm above the baseline simulating the pattern seen with acute MI, pericarditis or ventricular aneurysm. However these changes are stable, they do not undergo the evolutionary sequence seen in pericarditis and are not associated with reciprocal ST depressions as is observed in acute MI.

T wave inversion. T wave may be inverted in leads III and aVF and in chest leads V1 and V2 in normal individuals. An inverted T wave in leads I, II or V3 to V6 is usually abnormal.
If in doubt about the presence of acute myocardial ischaemia, repeat ECGs and correlate with the presence of chest pain and troponin rise.

Q. When may ECG signs be falsely positive for dextrocardia?

A. Limb lead reversal (not uncommon!). Reversal of the right and left arm electrodes will cause an apparent rightward QRS axis shift that can lead to an incorrect diagnosis of dextrocardia. As a general rule when lead I shows a negative P wave and QRS, suspect the right and left arm leads have been reversed.

Remember: Always interpret data and signs in context of the overall clinical picture.

Common ECG artefacts

- 60 Hz cycle interference produced by alternating current generators (switch the ECG plug to another outlet).
- Muscle tremor e.g. parkinsonism, shivering.
- Improper standardisation.

ECG ARTEFACT SECONDARY TO DIALYSIS (CVVHD) ROLLER PUMP
LIMB LEADS CORRECT

LIMB LEADS INCORRECT

ECG MOVEMENT ARTEFACT NOTE NORMAL ARTERIAL PRESSURE TRACE
Pulse oximetry

Although pulse oximetry is a valuable monitoring tool used throughout the hospital and associated with few complications, there is often a lack of understanding of what is being measured. The measurements are often inaccurate in the presence of alterations in skin perfusion. The fundamental principles of pulse oximetry are well explained in the references cited below.


**NOTE** The oxyhaemoglobin dissociation curve has a sigmoid shape. Near maximal (90-100%) oxygen saturation of haemoglobin occurs at a PaO2 of 8.0 kPa (60 mmHg). Values above this only produce a modest increase in SpO2. Conversely, oxygen saturation values below 90% may rapidly decrease further and are associated with low PaO2 levels.

**WARNING** Pulse oximetry reflects oxygenation of arterial blood. However, significant alveolar hypoventilation and hypercapnia can occur despite an unchanged SpO2, particularly if the patient is receiving supplemental oxygen.
A 70-year-old patient with past history of COPD and a recent flu vaccination is admitted to a medical ward with lower limb paraesthesia and weakness. Observations including heart rate, AP, respiratory rate and SpO₂ (2 L/min oxygen) are stable. However she is drowsy since admission and the intensive care team is asked to evaluate her when she cannot be roused. On examination she is unresponsive to painful stimuli and her extremities are cold and mottled. Heart rate 60 beats/min, AP 120/80 mmHg, respiratory rate 16/min and SpO₂ 95%. Her trachea is immediately intubated. An arterial blood gas reveals profound respiratory acidosis with pH 6.8, PCO₂ 20 kPa (150 mmHg), PaO₂ 14.0 kPa (105 mmHg), bicarbonate concentration 15.3 mmol/L, base excess -14.0 mmol/L and serum lactate 1.5 mmol/L. She is transferred to the intensive care unit and Guillain-Barré syndrome is subsequently diagnosed. Pulse oximetry measures the adequacy of arterial haemoglobin saturation, not the adequacy of ventilation.

Situations where the pulse oximeter will fail to detect true oxygen saturation of haemoglobin:

- Dyshaemoglobins. Carboxyhaemoglobin (COHb) produces an SpO₂ reading that includes both COHb and oxygenated haemoglobin (HbO₂), making the SpO₂ reading falsely elevated. Similarly, the presence of significant methaemoglobin markedly reduces the accuracy of oximetry.
- In low perfusion states, hypothermia, cardiac arrhythmias and when excessive motion is present the oximeter may fail to accurately differentiate true signal from background noise and thus may produce erroneous data.
- Dyes, e.g. methylene blue (used to treat methaemoglobin toxicity) can falsely lower SpO₂ reading.

Anaemia does not reduce the accuracy of the pulse oximeter provided that haematocrit remains >15%.

Venous oximetry

Decrease in SvO₂/ScvO₂ represents either an increase in oxygen consumption (exercise, pyrexia, increased work of breathing, shivering, pain) or decreased oxygen delivery (decreased arterial oxygen content, e.g. anaemia, hypoxia) or inadequate cardiac output e.g. hypovolaemia, heart failure.
Q. Explain how whole body oxygen consumption is measured.

A. Oxygen consumption (VO₂) is expressed mathematically by the Fick principle as the product of cardiac output (CO) and arteriovenous O₂ content difference (CaO₂ - CvO₂).

This may be rewritten as:

\[ VO₂ = CO \times (CaO₂ - CvO₂) \]

\[ CvO₂ = \frac{CaO₂ - VO₂}{CO} \]

CaO₂ = Arterial oxygen content = (Hb x 1.39 x SaO₂) + (0.003 x PaO₂)

CvO₂ = Mixed venous oxygen content (CvO₂) = (Hb x 1.39 x SvO₂) + (0.003 x PaO₂)

Therefore the equation may be rewritten as:

\[ (Hb \times 1.39 \times SaO₂) + (0.003 \times PaO₂) = (Hb \times 1.39 \times SvO₂) + (0.003 \times PaO₂) - \frac{VO₂}{CO} \]

Because at standard atmospheric pressure, the quantity of dissolved oxygen is very small, it is acceptable to eliminate this component and re-write the equation as:

\[ Hb \times 1.39 \times SvO₂ \neq Hb \times 1.39 \times SaO₂ - \frac{VO₂}{CO} \]

As SaO₂ is maintained at near maximal saturation in most patients and is not rapidly changing, the equation can be further reduced to:

\[ \Rightarrow SvO₂ \sim \frac{VO₂}{CO} \]

Thus SvO₂ is directly proportional to the following; SaO₂, the ratio of oxygen consumption to cardiac output and haemoglobin. Therefore SvO₂ reflects the relationship between O₂ consumption and oxygen delivery.

Q. Write the Fick equation and show how it is used to measure cardiac output.

A. The determination of oxygen saturation in mixed venous blood (SvO₂) enables interpretation of the cardiac output by considering oxygen transport in relation to oxygen consumption. From the Fick equation:

\[ VO₂ = CO \times (CaO₂ - CvO₂) \]

\[ VO₂ = CO \times Hb \times 1.39 \times (SaO₂ - SvO₂) \times 10 \]

\[ SvO₂ = \frac{SaO₂ - \frac{VO₂}{CO \times Hb \times 13.8}}{10} \]

Q. List three conditions associated with a low SvO₂/ScvO₂.

A. A low SvO₂ can reflect three situations:
- Hypoxaemia (a fall in SaO₂ causes a direct fall in SvO₂)
- Anaemia (with incomplete compensation by the cardiac output)
- An increase in the relationship between oxygen consumption \( (V_O₂) \) and cardiac output. In other words inadequate cardiac output in relation to the oxygen demand e.g. cardiac failure, pulmonary embolism, hypovolaemia.

**NOTE** During exercise, increased oxygen demands are met primarily by increasing cardiac output and only secondarily by increasing oxygen extraction. Thus SvO₂ may decrease somewhat during exercise but does not necessarily reflect tissue hypoxia.

**Relationship between \( S\overline{v}O₂ \) and \( ScvO₂ \)**

In health, \( S\overline{v}O₂ \) is 2-3% higher than \( ScvO₂ \) because the lower body extracts less oxygen than the upper body making the inferior vena cava saturation higher. The primary cause is that the kidneys and liver receive a high proportion of cardiac output but oxygen consumption is low relative to delivery.

In shock, this relationship changes and the \( ScvO₂ \) may exceed \( S\overline{v}O₂ \) values by up to 8%. This is because in shock states, splanchnic and renal circulation fall followed by an increase in \( O₂ \) extraction in these tissues. In septic shock, regional \( O₂ \) consumption of the gastrointestinal tract increases. On the other hand, flow to the heart and brain is maintained. Hence \( ScvO₂ \) is a less reliable guide for \( S\overline{v}O₂ \) in critically ill patients.

\( ScvO₂ \) should not be used alone in haemodynamic assessment but combined with other indicators of organ perfusion such as mental status, urinary output and serum lactate levels.

The evidence for targeting an \( ScvO₂ \) value of >70% as a treatment goal in septic shock comes from one study. The Rivers early goal-directed therapy study showed that \( ScvO₂ \) is useful in guiding the early resuscitation of septic shock using a target of \( ScvO₂ >70% \) during the first six hours of treatment.


Non-invasive monitoring of arterial blood pressure

Sources of error in taking non-invasive AP measurements include:

‘Auscultatory gap’: in some, especially older and hypertensive patients, the Korotkoff sounds may disappear and then reappear at a lower pressure. This can lead to underestimation of true systolic AP.

Optimum size of occluding cuff: the width of the bladder should be 40% of the circumference of the upper arm at the midpoint and its length should be twice the circumference of the upper arm.

Arm position: requires patients to be in a supine position when pressures are measured.

Complications, although rare have been reported during non-invasive AP measurement:

Ulnar nerve palsies have been reported with frequent inflation and deflation of upper arm AP cuff.

In patients with upper arm AV fistula for haemodialysis, cuff inflation may damage the fistula. In patients post mastectomy with extensive axillary clearance, upper limb oedema may develop with multiple cuff inflations. The AP cuff should be placed on the opposite arm in these instances.

Invasive pressure monitoring

It is useful to consider complications associated with invasive pressure monitoring in three categories:

1. During insertion or removal of the monitoring catheter
2. While the catheter is in situ
3. Inaccurate or incorrectly interpreted data obtained from catheter.

Complications during insertion and removal of monitoring catheters

Invasive arterial pressure catheters

Common to all sites: pain, haemorrhage or haematoma, (traumatic arterial puncture), nerve damage.

Individual sites: femoral artery site; retroperitoneal haematoma formation, bowel perforation.

Central venous catheter

Common to all sites: as above.
Arterial puncture and haemorrhage.

Intrathoracic sites: pneumothorax (subclavian site > internal jugular site).

Arrhythmias during guide wire passage through RV.

Perforation of mediastinal vessel or cardiac chamber.

Venous air embolism, especially if the patient is generating significant negative intrapleural pressure e.g. laboured breathing. In the presence of right-to-left shunt (e.g. ASD), air may cross to left heart and cerebral circulation.

**Pulmonary artery catheter**

*Common to all sites* (including intrathoracic sites): as above.

Ventricular ectopy is common. Sustained arrhythmias usually occur in conditions leading to myocardial irritability (e.g. electrolyte imbalance, acidosis, myocardial ischaemia). A defibrillator should be immediately available in the event of a sustained ventricular arrhythmia.

Right bundle branch block (RBBB) may be induced by PAC contact with the right side of interventricular septum - especially during insertion.

Catheter coiling or knotting typically occurs during prolonged and difficult passage across the pulmonary valve (e.g. dilated RV or low CO state) and is confirmed with chest X-ray. Removal often requires use of a fluoroscopy guided snare device.

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**Caution:** Traumatic vessel puncture during cannulation can cause substantial haemorrhage without overt evidence of a problem at the insertion site. For example, femoral artery/vein trauma causing massive retroperitoneal bleeding requires CT (computed tomography) scan to diagnose (not visible on ultrasound). Traumatic puncture of internal jugular or subclavian vein may be complicated by mediastinal bleeding or haemothorax visible only on chest X-ray.

**Caution:** Exercise caution during insertion of catheters with long dilators e.g. PAC sheath and dialysis catheters. The rigid dilator may perforate a cardiac chamber or central vessel. Dilators do not need to be inserted completely, since their purpose is to dilate the skin and the vessel puncture site.

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Use of bedside ultrasound, compared to use of standard anatomical landmarks, has been shown to reduce the risk of mechanical complications during insertion of central catheters.

If venous air embolism is suspected, place patient head down in left lateral decubitus position and aspirate the lumen of the venous catheter.

There is a small risk of precipitating complete heart block during insertion of PAC in patients with LBBB on ECG.
Complications occurring with monitoring devices in situ

Invasive arterial pressure monitoring

*Common to all sites:* catheter-related infection includes both insertion-site infection and catheter-related bloodstream infection (CRBSI). The majority of serious catheter-related infections are associated with central venous catheters. The infection rate is higher with catheters inserted in the internal jugular vein compared to the subclavian vein. Refer to the PACT module on Infection Prevention and Control.

Heparin-associated - thrombocytopenia.
Blood loss.
Pseudaneurysm formation (requires surgical repair as it may rupture or cause embolisation).
Thrombus formation in a patient with poor collateral flow may compromise circulation and risk distal ischaemia.

*Individual sites:* Femoral site insertion reduces mobility.

**Central venous catheter**

*Common to all sites:* as above.

Vascular erosion may occur 1-7 days following catheter insertion. Free aspiration of blood from the catheter does not rule out vascular perforation. Proper positioning of the catheter tip parallel to the vessel wall, should be checked post-insertion and on all chest X-rays.

Central venous thrombosis. This can lead to partial venous occlusion, and in a small number of patients total occlusion of a vein can occur. Total occlusion may be clinically identified by oedema of the involved arm, neck or face.

**Pulmonary artery catheter**

Rupture or perforation of a pulmonary artery may be manifested as sudden haemoptysis (or blood from the tracheal tube) or as an infiltrate on chest X-ray associated with the location of the catheter tip. Less commonly, PA perforation may cause haemothorax or cardiac tamponade.

Pulmonary infarction can occur secondary to vascular occlusion due to catheter or catheter-related thrombus or prolonged balloon inflation.

Intracardiac injury to valves or ventricular surface. Balloon rupture, manifest as blood return from balloon port may lead to air embolism if the balloon port is not tightly closed off to air and/or further attempts are made to inflate the balloon.

The most common in-situ complication of invasive monitoring devices is infection.

Central venous catheter vascular erosion is most common when the catheter is inserted from the left internal/external jugular or subclavian site as the tip is more likely to be against the wall of the superior vena cava.

Risk factors for perforation of the pulmonary artery by PAC include: catheter advanced or migrated too far distally, pulmonary hypertension, excessive manipulation or balloon inflation, and, hypothermia (catheter becomes stiffer).
Limiting the duration of PAC insertion, shortening duration of balloon inflation, continuous monitoring of waveform from the catheter tip and checking catheter position on chest X-ray reduce the incidence of PAC complications.

Central venous catheter-related complications are discussed in detail in the following references.


Complications related to incorrect collection or interpretation of data

Meticulous measurement technique and analysis of pressure waveforms, knowledge of the pitfalls for invalid measurement and an understanding of the relationship of pressure measurement to cardiovascular physiology are mandatory to ensure that haemodynamic data obtained are reliable and effectively used at the bedside.

Invasive arterial pressure monitoring

Common to all fluid-filled monitoring systems: the ability to accurately display measured pressure from the catheter tip depends on accurate transmission to an appropriately zeroed and calibrated monitor. If the zero point is not set correctly, measurements will be biased (consistently read high or low over the entire scale), known as an offset error. Refer to Task 2.


Central venous pressure

Patient-related factors: Acute or chronically decreased right ventricular compliance may result in disproportionately high pressure values for a given filling volume. Tricuspid valve disease (stenosis/regurgitation) increases the CVP; in tricuspid regurgitation the V wave becomes dominant and elevated, raising the mean pressure. Left-to-right intracardiac shunts (acute VSD) increase right ventricular volume and, thus, increase CVP.

Measuring the mean A wave is necessary to assess right ventricular filling pressure. Pericardial tamponade should also be considered.
Mechanical ventilation factors: CVP may be overestimated in patients receiving ventilatory support due to increased intrathoracic pressures surrounding the heart and great vessels. Refer to Task 2.

Elevated intra-abdominal pressure may artefactually elevate CVP measurement.

CVP has limited value in estimating LV preload.

For more information see the PACT module on Abdomen in acute/critical care medicine.

Pulmonary artery catheter

Catheter-related factors:
Overdamping decreases systolic pressure and increases diastolic pressure.

Catheter whip causes spike-like artefacts superimposed on the PAC waveform and may occur in a hyperdynamic circulation.

Catheter tip may slip back into the RV. The waveform will differ in morphology with a notable fall in diastolic pressure to near baseline (0). Ventricular ectopy may be noted on the ECG (see Task 2). If in doubt, chest X-ray will confirm the position of PAC tip.

Overwedging may occur if balloon inflates unevenly and herniates over the catheter tip or the distal lumen becomes blocked against a wall. The resultant waveform appears damped and lacks any recognisable PA or PAOP morphology and the pressure gradually equilibrates with that in the flush system.

Persistent wedging may occur if the catheter tip migrates distally into a small segment of the PA. This is more likely to occur with excessive catheter looping in the RV during insertion. Continuous monitoring of the pressure from the catheter tip is essential to recognise this occurrence.

⚠️ Correct recognition of PAOP waveform requires experience. Always discuss your findings with an experienced colleague. PAOP should not be interpreted in isolation but in the context of clinical findings.

In the next ten patients with a PAC in situ, measure PAOP and then ask the bedside nurse to do the same. Now discuss the findings with the ICU consultant.

There is large interobserver variability in interpretation of the PAOP waveform.
Limitations during measurement of PAOP

Patient-related factors: The relationship between PAOP, left atrial pressure and left ventricular end-diastolic pressure may not be close in certain situations.

Giant ‘V’ waves in the PAOP are often associated with acute mitral regurgitation due to retrograde blood flow into the left atrium during ventricular systole. A simultaneously recorded ECG and PAOP trace most reliably confirms a giant V wave. The V wave will be located later in the cardiac cycle (well after the T wave) than pulmonary artery systolic wave. The presence of a large V wave will cause the displayed mean PAOP to be higher than the pulmonary artery diastolic pressure and the mean pressure displayed may change minimally with balloon inflation. Accurate measurement of the PAOP as a reflection of the LV filling pressure requires measurement of the average height of the A wave of the PAOP at end-expiration. The A wave is located at the end of the QRS complex of the simultaneously obtained ECG.

The PAOP V wave may also be slightly dominant and elevated with reduced left atrial compliance. Giant V waves may be due to decreased left ventricular compliance or hypervolaemia.

Large, or cannon A waves in the PAOP may be seen in patients with A-V dissociation, when atrial contraction occurs at the time of ventricular contraction. The presence of cannon A waves elevates the PAOP and is not reflective of left ventricular end-diastolic pressure.

Failure to identify a large V wave on a PAC waveform may create an impression that the catheter has failed to wedge, resulting in repeat attempts to wedge and possible pulmonary artery damage. Careful inspection of the waveform in relation to the ECG is essential to prevent this occurrence.

Mitral stenosis or left atrial myxoma prevents diastolic equilibration of pressures between the LA and the LV and hence neither the PA diastolic pressure nor the PAOP reflect the LVEDP.

Tricuspid regurgitation may cause difficulty in advancing the PAC across the tricuspid valve. In addition, it interferes with the reliable estimate of CO by thermodilution leading to variability in the signal which may lead to under or overestimation of CO. Tricuspid regurgitation also produces large V waves in the RAP or CVP. Accurate reflection of RVEDP requires measurement of the average height of the end-expiratory RA or CVP A wave measured in the T-P interval of the simultaneously obtained ECG.

Intracardiac shunts interfere with determination of thermodilution CO by causing recirculation of cold injectate (right-to-left) or inadequate mixing (left-to-right).
Left ventricular end-diastolic volume (LVEDV) is the most accurate clinical measure of LV preload. The PAC measures PAOP which is an estimate of LVEDP. However, there must be a linear and predictable relationship between LVEDP and LVEDV in order for the PAOP to be a reliable indicator of LV preload. Any change in LV compliance uncouples this pressure/volume relationship.

In critically ill patients many of the factors that determine LV compliance are in a state of dynamic flux making it very difficult to estimate LVEDV from LVEDP.

Mechanical ventilation factors: increased intrathoracic pressure associated with PEEP has a significant effect on juxtacardiac pressure. This effect may artefactually increase PAOP. See Task 2.

Limitations of pulse contour analysis and transpulmonary thermodilution

- A reliable arterial waveform trace is essential for accurate cardiac output measurements. Over- or under-damping of the arterial waveform will lead to incorrect measurements of cardiac output.
- Intra-aortic balloon pump alters the arterial waveform.
- Arrhythmias, e.g. atrial fibrillation.

Minimally invasive methods of measuring cardiac output and cardiac contractility

The new methods that challenge the PAC for measuring cardiac output are reviewed in the following reference.
Stroke volume/cardiac output/cardiac contractility

Measurement of stroke volume (or stroke index) or cardiac output (or cardiac index) and cardiac contractility are useful for differentiating the causes of haemodynamic shock according to traditional classifications: hypovolaemic, cardiogenic, sepsis (distributive) and obstructive (pulmonary embolism, dissecting aneurysm, pericardial tamponade). This classification has practical merits useful for treatment while recognising that it oversimplifies the pathophysiology of shock.

The ‘normal’ range of stroke index is 25-45 mL/beat m² and 2.5-3.5 L/min/m² for cardiac index (cardiac output/body surface area). However, less importance should be placed on a particular number and more on the combination of clinical examination and haemodynamic data for an individual patient. Stroke volume/index or cardiac output/index is adequate if there is no evidence of tissue hypoperfusion.

For more information see the PACT modules on Heart failure, Acute myocardial ischaemia and Hypotension.

The relationship between cardiac output, myocardial contractility and S\textsubscript{v}O\textsubscript{2} in haemodynamic shock of different aetiologies is shown in the following table. The changes in cardiac output are mirrored by changes in ScvO\textsubscript{2} (or SvO\textsubscript{2}). In septic shock, although cardiac output is elevated, ejection fraction is frequently decreased.

<table>
<thead>
<tr>
<th>Aetiology of shock</th>
<th>Cardiac output</th>
<th>Ejection fraction</th>
<th>S\textsubscript{v}O\textsubscript{2}</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolaemia</td>
<td>↓</td>
<td>normal/↑</td>
<td>↓</td>
</tr>
<tr>
<td>Cardiogenic</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Sepsis</td>
<td>normal/↑</td>
<td>↓</td>
<td>normal/↑</td>
</tr>
<tr>
<td>Obstructive</td>
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In critically ill patients, there may be overlap of signs and symptoms between the different categories of shock. Data from the SHOCK trial indicated that 18% of patients with cardiogenic shock following MI were also suspected of having sepsis as a cause of shock. Keep an open mind when interpreting haemodynamic data.


Pulmonary artery catheter

The indications for insertion of a pulmonary artery catheter (PAC) are discussed in Task 1. It is useful to consider the haemodynamic data obtained from the PAC in the form of a schematic representation of the heart.

NOTE Vascular resistance is expressed as metric units (dynes/sec/cm\(^{-5}\)) or resistance units (mmHg/L/min). The difference between them is that metric units are greater by a factor of 80. Resistance units are also called Wood units after the cardiologist who introduced them.

Q. What are the normal oxygen saturation values encountered during right heart catheterisation?

A. Approximate normal oxygen saturation values during right heart catheterisation:
   - Superior vena cava: 70%
   - Right atrium: 75%
   - Right ventricle: 75%
   - Pulmonary artery: 75%
   - Wedged position, blood aspirated from distal port: 98%
**Haemodynamic data from the PAC in different clinical scenarios**

**Cardiogenic shock**: clinical signs of hypoperfusion in conjunction with haemodynamic data demonstrating decreased cardiac index and markedly elevated PAOP.

**CARDIOGENIC SHOCK**

Right heart failure: increases in RV end-diastolic and mean RA pressures. If caused by LV failure, these increases will be accompanied by an increase in PAOP and a decreased cardiac index. In the setting of an isolated RV infarct, the RA pressures will be disproportionately elevated compared to PAOP.

**RIGHT VENTRICULAR INFARCT CARDIOGENIC SHOCK**
**Septic shock:** in the early stages, in particular, low arterial pressure, normal or low PAOP and increased stroke index/cardiac index may be manifest. LV failure may occur in the later stages of the disease.

**SEPTIC SHOCK**

![Diagram of septic shock](image)

**Ventricular septal defect:** Acute ventricular septal rupture: right heart catheterisation may show a step-up in oxygen saturations of blood (>7%) in the pulmonary artery compared to the right atrium.

**ACUTE VENTRICULAR SEPTAL DEFECT**

![Diagram of ventricular septal defect](image)
**Acute mitral regurgitation:** in addition to a systolic murmur, a giant V wave occurs in the PAOP waveform. With a simultaneously recorded ECG, the PAOP V wave of acute mitral regurgitation occurs (well after the T wave), while the peak PA systolic wave (occurs within the QRS). See Task 2.

‘**V’ WAVE PCWP**

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**Acute massive pulmonary embolism:** moderate pulmonary hypertension is seen in acute pulmonary embolism. However, the RV and PA systolic pressures rarely exceed 50-55 mmHg. At greater pressures in the acute setting the RV dilates and can fail. The finding of a pulmonary artery systolic pressure >55 mmHg indicates chronicity as the thin walled RV requires time to hypertrophy to generate the higher pressure. The RA pressure is elevated and greater than the PAOP which is usually normal or low.

**ACUTE MASSIVE PULMONARY EMBOLISM (PE)**

Cardiac index: <2.0
**Cardiac tamponade**: the haemodynamic hallmark is equalisation of the RA, RV and PA diastolic and PAOP pressures.

**CONCLUSION**

This module has highlighted how haemodynamic monitoring is used to assess global and regional tissue perfusion at the bedside in critically ill patients. Fundamental to the use of haemodynamic monitoring is the principle that changes in outcome depend on the treatment changes that are guided by the information obtained, rather than on the monitors per se. The process is patient - rather than technology - centred and repeated clinical assessment is an integral part of haemodynamic monitoring. The module is designed to reflect the clinical environment where monitoring and clinical assessment are used in a stepwise fashion. The least invasive monitoring system that can aid diagnosis and usefully guide treatment is the system of choice. It is strongly recommended to assess changes in monitored values over time and in response to treatment, rather than single values.
**PATIENT CHALLENGES**

Patient 1

A 68-year-old obese (100 kg) male is admitted to the Emergency Department after a road traffic accident. He is short of breath and complains of pain during breathing. Observations include respiratory rate 30/min, pulse 110/min, blood pressure (BP) 130/70 mmHg and SpO₂ 92%. Initial surveys and X-rays reveal sternal bruising, left-sided rib fractures, a compound left supracondylar fracture, a left femoral fracture and pelvic fractures. The past medical history is significant for hypertension and myocardial infarction.

http://www.facs.org/trauma/atls/index.html

**Learning Issues**

PACT module on Multiple trauma

Q. Presuming pain relief has been adequately addressed, how would you interpret the haemodynamic data?

A. Tachycardia and tachypnoea in this setting may indicate the presence of compensated haemodynamic shock. Given the history of hypertension, the BP may be lower than normal for this patient and should be interpreted in context.

**NOTE** Pain may mask hypovolaemia and a decrease in blood pressure may be a late sign of severe blood loss.

The spine X-ray series show no bony injury. A CT (computed tomography) scan of the chest shows a normal aorta, bilateral lung contusions, a small left haemothorax and no pneumothorax. The liver, spleen and kidneys are intact.

The O₂ saturation (SpO₂) drops while the patient is supine in the CT scanner and a pelvic scan is abandoned. Despite increases in FiO₂ the patient remains hypoxic and in increasing respiratory distress and the decision is made to intubate the trachea.

Q. What risk to the circulation is posed by intubation and mechanical ventilation in this patient?

A. Hypotension.
Q. Give three possible mechanisms for the hypotension.

A.

1. Hypovolaemia may be unmasked (perhaps dramatically) when a patient is sedated as increased sympathetic tone (due to pain and anxiety) may have been causing the blood pressure to be maintained.

2. Sedative/anaesthetic drugs may have a direct cardiodepressant and/or vasodilatory effect.

3. Positive intrathoracic pressure from mechanical ventilation will also decrease venous return and cardiac output.

During intubation, additional fluids are given to treat hypotension. On arrival in the ICU, the nurse attaches a pulse oximeter to the left hand but is unable to get a signal. When the device is placed on the right hand there is a low amplitude waveform.

Blood pressure is 85/60 mmHg and heart rate 120 beats/min.

Q. Monitoring problems might be patient- or device-related. What patient factors might have compromised the signal from the pulse oximeter on the left hand?

A. A patient factor may have been the left supracondylar fracture compromising the brachial artery. (The CT scan ruled out aortic pathology). There is also likely to be a systemic problem (hypovolaemia) as evidenced by the poor signal on the right side.

Learning Issues

When haemodynamic monitoring data is inconsistent, re-examine the patient


Q. If you suspected a device-related factor, how would you check for this?

A. Make sure that the probe, connections and set-up of the monitor are correct. You can check device-related problems quickly by connecting the probe to your own finger.

Learning Issues

Trouble-shooting a defective oximetry trace entails checking the device

Principles of pulse oximetry
Q. If there was clinical evidence of brachial artery compromise, what would the priority be now?

A. Decompression/revascularisation of the left upper limb. Urgent consultation to the vascular and orthopaedic surgeons is required.

Manipulation of the supracondylar fracture under general anaesthesia achieves reperfusion of the left hand. Following internal fixation of the femoral fracture the patient returns to the ICU. Hypotension, arterial pressure (AP) 85/45 mmHg, is a problem despite additional fluid therapy perhaps because of covert bleeding from pelvic fractures.

Q. What types of haemodynamic monitoring might you consider?

A. The current state suggests hypovolaemia and you institute invasive AP (right-sided, given the left arm injury) and central venous pressure (CVP) monitoring.

Learning Issues

Indications for invasive haemodynamic monitoring

Q. What are the advantages of invasive AP monitoring at this stage?

A. Beat-to-beat AP and monitoring of pulse pressure variation and it allows serial measurements of gas exchange by blood gas analyses.

Q. What are the advantages of CVP monitoring at this stage?

A. The CVP response to fluid challenges is a useful guide to resuscitative therapy. In addition, the AP/CVP combination allows estimations of global tissue perfusion by serial lactate and ScvO₂.

Q. Is a pulmonary artery catheter warranted?

A. A pulmonary artery catheter (PAC) is not indicated during initial resuscitation.

Learning Issues

Pulse pressure variation
Markers of global tissue perfusion
PAC is not indicated during initial resuscitation

Despite further fluid resuscitation, the patient remains hypotensive, with a lactate level of 5.4 mmol/L and ScvO₂ of 58%.
Q. Other than hypovolaemia, what are the other e.g. cardiopulmonary possible causes of hypotension and global malperfusion?

A. The sternal bruising may be associated with cardiac contusions or pericardial effusion. Pulmonary contusions may cause pulmonary hypertension (hypoxic pulmonary vasoconstriction), increasing right ventricular afterload and contributing to right heart failure. There may be underlying ventricular dysfunction from a previous (or indeed an acute) myocardial infarction.

Q. If CVP is low, does this help to differentiate between the above mentioned possible causes of the patient’s hypotension?

A. A low CVP means right heart contusion, tension pneumothorax and tamponade are unlikely. Although a low CVP is suggestive of hypovolaemia, remember that hypovolaemia may not be the only clinical problem as heart failure may co-exist with hypovolaemia in the presence of the low CVP.

When measured, the CVP was 14 mmHg.

Q. What simple intervention might you use to elucidate the nature of shock present?

A. Administer a fluid challenge and assess the patient response. See Task 1.

Q. What additional measures or monitoring could you use to establish whether heart failure is the problem?

A. Echocardiography would enable assessment of right and left ventricular function, filling status of the ventricles and rule out pericardial effusion. A pulmonary artery catheter would give information on right- and left-sided filling pressures and cardiac output (CO).

NOTE: Echocardiography is the test of choice in a hypotensive trauma patient. Pericardial effusion and tamponade can be demonstrated. With cardiac contusion there may be a reduction in ejection fraction and wall motion abnormalities may be present.

Learning Issues
Diagnosis of heart failure using a pulmonary artery catheter

Because of ongoing poor oxygenation and cardiovascular impairment, you insert a pulmonary artery catheter that displays continuous cardiac output and SvO₂. Body surface area measures 2.5 m². Haemodynamic data:
- Heart rate 110 beats/min
- AP 90/55 mmHg
- SaO₂ 95%
- Serum lactate 5.8 mmol/L
- Urinary output 5-10 mL during the last three hours
- Haemoglobin 7.0 g/dL

Q. How do you interpret the pressure data with respect to right and left heart function?

A. The moderate pulmonary hypertension is related to pulmonary contusion and not to left ventricular (LV) failure as PAOP is normal. The diastolic pulmonary artery pressure to PAOP gradient indicates increased pulmonary artery resistance secondary to acute lung injury. Right atrial pressure is higher than PAOP indicating right ventricular (RV) dysfunction due to a combination of RV contusion and pulmonary hypertension.


**NOTE**

1. A gradient >5 mmHg between pulmonary artery diastolic pressure and PAOP indicates increased pulmonary artery resistance.
2. Normally PAOP is higher than RAP by 2-3 mmHg, the reverse indicates RV dysfunction or pulmonary vasoconstriction.
The ICM consultant performs a transoesophageal echocardiogram.

**Learning Issues**
Echo diagnosis of right heart failure

Q. Are the echo findings consistent with the PAC data? Give your reasons.
A. Yes. There is RV hypokinesis. There is no evidence of cardiac tamponade.

Q. Is the cardiac output adequate in this patient? Give reasons.
A. No. The low $\text{SvO}_2$ and elevated serum lactate confirm global tissue hypoperfusion. The cardiac index ($\text{CO} / \text{BSA}$) of 2.0 L/min/m² is below normal range. A low CI combined with tachycardia indicates a low stroke volume index; in this case 16.7 mL/m².

**Learning Issues**
Calculation of derived haemodynamic variables from pulmonary artery catheter

To increase CO and oxygen delivery, you ask the nurse to perform a fluid challenge (300 mL of packed red blood cells over 30 min).

You provide a simple decision rule for the nurse to terminate the fluid challenge.

An acute and consistent increase in either RAP or PAOP without a concomitant increase in stroke index indicates ventricular overload and fluid resuscitation should be stopped.

Following three consecutive fluid challenges, the haemodynamic assessment shows:
- Heart rate 95 beats/min
- AP 95/60 mmHg
- SaO₂ 95%
- Serum (blood) lactate 4.5 mmol/L
- Urine output 65 mL during the last two hours
- Haemoglobin 9.0 g/dL

**NOTE:** Continuous cardiac output monitoring facilitates assessment of a fluid challenge.

**Q.** The stroke volume index has increased (now 28 mL/m²). Do you consider the overall changes in values to meet the goals of your fluid challenge? Explain your answer with reference to the therapeutic goals you were using.

**A.** Yes. Your goal was to increase CO by optimising stroke volume and thus improve oxygen delivery. This was associated with an increase in SvO₂ and a decrease in serum lactate. Urinary output has also improved, indicating improved perfusion to the kidneys.
Q. If the SvO₂ remained below normal and serum lactate continued to rise, indicate two further pharmacologic interventions you could consider to improve cardiac output.

A. 

1. Administration of dobutamine would increase stroke volume and cardiac output (β₁-agonist action) and decrease pulmonary vascular resistance.

2. A pulmonary vasodilator such as inhaled nitric oxide might be considered to lower pulmonary artery pressure and, by reducing RV afterload, lead to an improvement in right ventricular function.

Q. Explain the reversal of the normal relationship between ScvO₂ and SvO₂.

A. There is a reduction in blood flow to the splanchnic and renal circulation, resulting in a fall in oxygen content in the inferior vena cava. Therefore ScvO₂, which is measured from the SVC, is greater than SvO₂. See Task 2.

Learning Issues

ScvO₂ monitoring

ScvO₂ may be greater than SvO₂ in shock states

PACT module on Heart failure


The patient is haemodynamically stable. However, 24 hours later, on clinical examination you auscultate a new pansystolic murmur. There is an apical systolic thrill.

Q. Can the central venous and pulmonary artery catheters help with diagnosing the cause of the murmur? Explain your answer.

A. Yes. The possible causes of pansystolic murmur in this setting are tricuspid regurgitation, mitral regurgitation and ventricular septal defect. The central venous and pulmonary artery catheters may help differentiate between these possibilities.
The following RAP trace is recorded from the PAC.

Q. In this context, how do you interpret the RAP trace recorded from the PAC?
A. There is no evidence of a large V wave. Therefore tricuspid regurgitation is unlikely.

Learning Issues
A large V wave on a CVP trace may indicate tricuspid regurgitation

The following PAOP trace is obtained.

Q. In this context how do you interpret the PAOP trace?
A. There is no evidence of a large V wave. Therefore mitral regurgitation is unlikely. In addition, the relative haemodynamic stability makes acute mitral regurgitation unlikely.

Learning Issues
A large V wave on a PAOP trace may indicate mitral regurgitation

The PAC shows the following O₂ saturations.
Q. In this context how do you interpret the O₂ saturations obtained from the PAC?

A. There is step-up in O₂ saturation of >8% between the right atrium and pulmonary artery. This indicates a left to right shunt at the level of the right ventricle. The cause is a traumatic ventricular septal defect (VSD). The apical thrill on clinical examination is very suggestive of a VSD.

**NOTE** A step-up in O₂ saturation >8% in the chamber before and after an intracardiac site indicates a left to right shunt.

Q. How would you confirm the diagnosis of VSD?

A. Echocardiography is the haemodynamic monitor/diagnostic mode of choice to diagnose a VSD.

**Learning Issues**

**Echocardiography**

Echocardiography demonstrates dilated RV cavity with RV free wall hypokinesis (consistent with contusions), and an apical traumatic VSD. Blood pressure begins to fall. An intra-aortic balloon pump (IABP) is inserted as a bridge to surgery. Coronary angiography demonstrates no critical coronary artery stenosis. The patient undergoes surgical closure of a traumatic VSD. The postoperative course is relatively smooth; the IABP is removed day one and the patient extubated day five. He is subsequently discharged from hospital 23 days post admission.
Patient 2

At 01.00 hours, you are called to the haematology ward to see a 20-year-old pregnant (18/40 gestation) patient who is tachycardic (heart rate 140/min) and tachypnoeic. She has non-Hodgkin’s lymphoma, and abdominal lymphadenopathy has caused obstructive uropathy. Ten hours previously, bilateral nephrostomy tubes were inserted emergently. She is day eight post chemotherapy with cyclophosphamide, doxorubicin, vincristine and prednisolone. You notice that the patient is cold and ‘shut down’.

Q. What is your initial management?

A. High flow oxygen via face mask. You obtain a portable monitor displaying ECG, blood pressure and oxygen saturation (SpO₂).

Although the case is complex, this first line approach is the same in all critically ill patients. The non-invasive blood pressure (NIBP) measures 100/40 mmHg and the SpO₂ 94% (8 L/min O₂). You transfer the patient to the ICU.

Learning Issues

Oxygen and basic monitoring is the first line monitoring approach in all critically ill patients.

The referring resident tells you the patient has sickle cell disease (compound heterozygote) and a lower limb deep venous thrombosis, for which she is receiving low molecular weight heparin. On arrival in the ICU, a radial arterial catheter is inserted demonstrating AP 85/35 mmHg and an arterial blood gas shows a pH of 7.20, PaO₂ 4.5 kPa, and serum lactate 6.3 mmol/L.

See the PACT module on Bleeding and thrombosis

Q. How do you interpret the monitored data?

A. The patient is hypotensive with evidence of global tissue hypoperfusion.

See the PACT module on Hypotension

Q. Given her recent procedure (nephrostomy tube insertion), what is the most likely diagnosis?

A. Severe sepsis and sepsis-associated tissue hypoperfusion. Septic shock needs to be ruled out.
Q. Is there a background predisposing factor for sepsis in this patient? What is the likely pathological process and category of pathogen?
A. She is an immunosuppressed patient (post chemotherapy). She likely has a Gram-negative bacteraemia.

Q. Outline two other aetiological possibilities?
A.
1. Hypovolaemia secondary to a retroperitoneal bleed; the procedure was performed while the patient was anticoagulated.
2. Pulmonary embolism (PE) is possible despite anticoagulation. Sickle cell disease and malignancy create a prothrombotic tendency.

More than one pathological process may be present; keep an open mind when interpreting haemodynamic data

PACT module on Sepsis and MODS

PACT module on Immunocompromised patients

Treatment is commenced with fluid loading, normal saline (1,500 mL) being chosen in this instance. Blood pressure and heart rate respond. Haemoglobin measures 10 g/dL.

Following blood and urine cultures, i.v. piperacillin/tazobactam and gentamicin are commenced (safe in pregnancy). The patient becomes increasingly drowsy and is intubated and mechanically ventilated for airway protection. The AP drops to 80/35 mmHg, a central venous catheter is inserted and a noradrenaline infusion (0.1 mcg/kg/min) commenced. Haemodynamic data after the fluid therapy and the commencement of noradrenaline are:

- Heart rate 98 beats/min
- AP 90/50 mmHg
- CVP 25 mmHg
- SaO₂ 98%
- ScvO₂ 58%
- Serum lactate 7 mmol/L
- Urinary output 0-5 mL during the last 2 hours
Begin empiric antibiotic treatment (after cultures taken) while instituting haemodynamic treatment/monitoring.

Q. How do you interpret the haemodynamic data?
A. The circulatory hypoperfusion (lactate 7 mmol/L) and the failure of the hypotension to respond to fluid resuscitation defines (septic) shock.

Q. The elevated CVP is an unexpected finding. List a minimum of four possible reasons for this finding in this patient.
A. 
- The measurement may be erroneous
- Right ± left ventricular failure secondary to severe sepsis or chemotherapeutic drugs
- Pulmonary embolism
- Tension pneumothorax post central catheter insertion in a ventilated patient
- Cardiac tamponade due to malignant pericardial effusion
- Constrictive pericarditis

Assessment of an unexpected result involves rechecking the accuracy of the data.

You re-zero the CVP and check the transducer position; the measurement is 22 mmHg. The chest X-ray shows no evidence of pneumothorax. After discussion with the ICM consultant/colleague, an echocardiogram is performed to rule out cardiac tamponade. It shows very poor biventricular function, ejection fraction 20-25%.

A dobutamine infusion is commenced and gradually increased to 7.5 mcg/kg/min. Continuous veno-venous haemodialysis (CVVHD) is started as the serum potassium measures over 6.0 mmol/L. After three hours, the haemodynamic data are:

- Heart rate 90 beats/min
- AP 100/40 mmHg
- CVP 18 mmHg
- SaO₂ 98%
- ScvO₂ 67%
- Serum lactate 5 mmol/L
- Urinary output 0 mL

**NOTE** Cardiac tamponade, if suspected in a shocked patient, needs to be urgently ruled out.

Q. Is the cardiac output (CO) improving or adequate?

A. An increased ScvO₂ and falling serum lactate suggest an adequate CO. Urinary output would not necessarily increase due to a likely already established acute renal failure (despite relief of the urinary tract obstruction) and commencement of CVVHD.

**Learning Issues**

Assessing the adequacy of cardiac output

Blood cultures subsequently confirm Gram-negative bacteraemia and the patient is improving with treatment.

On day three however, a miscarriage is associated with massive blood loss (>3 litres). Low molecular weight heparin is ceased. Despite blood product transfusion and increasing inotrope support, hypotension (AP 80/40 mmHg) is a problem. The ICM consultant decides to insert a pulmonary artery catheter when some stability has been achieved. Haemodynamic data:

- Heart rate 120 beats/min
- SpO₂ 97%
- Serum lactate 6 mmol/L
Q. When the PAOP was being measured, there was concern that the trace was incompletely ‘wedged’. What is your assessment of the PAOP obtained?

A. The pulmonary artery diastolic pressure is less than PAOP and therefore is likely an erroneous measurement.

Learning Issues
PAOP is usually 2-3 mmHg lower than pulmonary artery diastolic pressure

Common problems with pulmonary artery catheter waveforms

You reposition the pulmonary artery catheter and measure PAP; 45/18 mmHg and PAOP 13 mmHg.

Q. Other than checking the chest X-ray, what additional check could you use to ensure the validity of the PAOP reading?

A. Aspiration of highly oxygenated (‘arterialised’) blood, when the balloon is inflated and the catheter wedged, is confirmation of the direct contact of the catheter tip with the pulmonary capillary bed and therefore of the validity of the recorded PAOP.
**Q. What therapeutic intervention would you make in this patient on obtaining the accurate PAOP (which measures 13 mmHg)?**

A. A fluid challenge. A PAOP of 13 mmHg in the presence of LV dysfunction may be inadequate.

The patient responds to further fluid loading with an increase in AP, CI and ScvO₂ and a fall in serum lactate. However the gas exchange deteriorates in association with radiological changes of pulmonary oedema. The inspiratory fraction of oxygen (FiO₂) is increased to maintain SaO₂ >94% and PEEP is set at 10 mmHg. Tidal volume (450 mL) and respiratory rate (15 mL/min) result in an inspiratory peak pressure of 38 mmHg and a plateau pressure of 30 mmHg in this patient.

**Q. What effect would the increase in PEEP have on the PAOP measurement?**

A. Positive juxtacardiac pressure at end-expiration (PEEP) will cause the measured PAOP to overestimate transmural pressure, thus measured PAOP will overestimate left ventricular filling pressure. Less compliant lungs will minimise the extent of this artefact.

Bleeding resolves and the patient becomes haemodynamically stable. During the next 48 hours excess fluid is gently removed via CVVHD. The patient is successfully extubated day five post admission and renal function begins to improve. A second cycle of chemotherapy was scheduled one month post discharge from ICU.
**Patient 3**

A junior doctor requests that you review a 45-year-old man with known alcoholic liver disease, admitted 24 hours previously following a seizure. His past medical history includes oesophageal varices, ischaemic heart disease, and COPD. On initial assessment he is jaundiced, has clinical evidence of ascites, and his level of consciousness is reduced (he localises to a painful stimulus, mumbles incomprehensible sounds, and opens eyes to pain). Despite oxygen therapy at 4 L/min via nasal prongs, his SpO2 reads 85%. He is cold and clammy to touch. Non-invasive systolic blood pressure reads 70 mmHg and his heart rate is 120 beats/minute.

**Q. What is your first step?**

A. You immediately increase his oxygen to high flow (10 L/min). While performing further assessment, you commence a fluid bolus with 500 mL crystalloid/colloid.

**Q. How would you interpret the clinical information given?**

A. This patient has evidence of tissue hypoperfusion and is shocked.

**Q. Why might his SpO2 be low?**

A. The low oxygen saturation reading could be due to hypoxia from respiratory failure or could be secondary to malperfusion.

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**Learning Issues**

Initial approach to a critically ill patient necessitates rapid resuscitation measures in parallel with clinical assessment.

Further clinical examination reveals that he is tachypnoeic (RR 24) and has bilateral crepitations on auscultation. On exposing his lower limbs you note mottling around the knees. Arterial blood gas measurements:

- PaO2 8.7 kPa (65 mmHg)
- PaCO2 4.7 kPa (35 mmHg)
- pH 7.25
- Lactate 9 mmol/L
- Base excess -7

**Learning Issues**

Clinical assessment, basic monitoring and assessment of global perfusion
Q. What is the cause of shock in this case?

A. Sepsis is likely, but further clinical information is required to determine the exact cause. You should keep an open mind at this stage.

This patient is at high risk for spontaneous bacterial peritonitis, as well as pneumonia, and CNS infection. Differential and additional diagnoses should include hypovolaemic shock from upper GI bleeding, cardiogenic shock following a possible recent cardiac event, acute decompensated liver failure with hepatic encephalopathy.

Learning Issues
Remember, more than one cause of haemodynamic shock may exist at the same time. Irrespective of the underlying cause, the initial steps in resuscitation will be similar.

PACT module on Heart failure
PACT module on Acute hepatic failure
PACT module on Sepsis and MODS

Q. What is your next step?

A. In the setting of respiratory failure and haemodynamic shock, along with reduced level of consciousness, emergent intubation and mechanical ventilation is mandated.

Q. How might intubation be approached? Name a potential hazard?

A. It should be approached with care as shock is already present and circulatory collapse could occur with sedative drug administration.

Q. Where might it be done?

A. The patient should ideally be transferred to the intensive care setting for this, if time and resources permit.

PACT module on Acute respiratory failure

You intubate the patient in ICU, and continue fluid resuscitation while nursing staff prepare arterial and central venous catheters to site. After taking blood cultures you administer broad spectrum antibiotics to cover possible aspiration pneumonitis and spontaneous bacterial peritonitis, and meningo-encephalitis. The septic work-up also included a peritoneal fluid tap but not a sample for CSF examination due to the bleeding risk - see below. You also commence a vasopressor infusion. Lab haematology values are:
PACT module on Severe infection

Q. What are the considerations in relation to insertion of invasive catheters?

A. There is an increased risk of bleeding complications during insertion of invasive catheters. In some cases it may be appropriate to transfuse platelets and plasma prior to line insertion, but the severity of the situation may not permit a delay while blood products are being prepared.

In this case you elect to proceed, choosing the femoral route and using realtime ultrasound to guide you.

Learning Issues

How do I set-up the chosen types of haemodynamic monitoring?

Complications of haemodynamic monitoring

The patient remains hypotensive despite 3L crystalloid fluid resuscitation and noradrenaline infusion at 0.2 mcg/kg/min.

Q. CVP reads 10 mmHg. How do you interpret this result?

A. A single CVP reading should not be interpreted in isolation.

Q. As part of this assessment, you wish to ascertain whether tissue perfusion has improved following your resuscitative measures. What will you ask yourself and consider?

A. Have capillary refill and skin perfusion improved? Is serum lactate reduced? Is urine output adequate? If not, further fluid resuscitation may be required.

NOTE All static measures of preload must be interpreted within the clinical context.

Q. If the response to your interventive management is inadequate at this stage, would you initiate further haemodynamic monitoring?

A. Yes.
Q. Why?

A. There is a risk of pulmonary oedema with excess volume loading. You want to ascertain if the patient remains fluid responsive and what is the cardiac output. Given the history of cardiac disease you also want to determine the cardiac contractility.

You perform bedside trans-thoracic echo. Windows are limited but you can clearly see a hyperdynamic heart with excellent contractility. You also note that the left ventricular walls are ‘kissing’ at end-systole.

Learning Issues

Echocardiography excludes a ‘cardiogenic’ component of haemodynamic shock. An ‘empty’ LV suggests that further fluid administration may be warranted.

You give a further challenge and also decide to use a PiCCO™ monitor to guide your further management. Arterial waveform analysis and thermodilution values obtained:

- PPV (pulse pressure variation): 18%
- GEDI (global end-diastolic volume index): 500 mL/ m²
- EVLWI (extravascular lung water index): 5 mL/kg
- PVPI (pulmonary vascular permeability index): 2

Q. How do you interpret these values, particularly the PPV of 18%?

A. In order to reliably interpret the PPV data you first confirm that the patient is not breathing spontaneously, that the tidal volume is ≥8 mL/kg, and that sinus rhythm is present. A PPV value of 18% indicates that fluid challenging should improve cardiac output. The thermodilution data support this.

Learning Issues

Interpreting dynamic measures of preload

PPV of ≥13% in septic patients has been shown to be a specific and sensitive indicator of preload responsiveness.

NOTE Prerequisites for the use of PPV include sinus rhythm, absence of spontaneous ventilatory effort and tidal volume ≥8 mL/kg.

Q. You obtain a portable chest X-ray, What further diagnostic tests would you consider at this point? Would you consider an upper GI endoscopy?

A. After discussion of the advisability of a CT scan of brain +/- abdomen, you agree on a CT brain and abdominal ultrasound. You decide against upper GI endoscopy at present as Hb has not dropped from previous levels, and there has been no clinical evidence of an upper GI bleed.
CXR shows right lower lobe pneumonia. Ascitic fluid polymorphonuclear count (100/mm³) is not indicative of spontaneous bacterial peritonitis (SBP) - see Learning Issue. CT brain reveals atrophic change but no evidence of raised ICP or intracerebral haemorrhage. Abdominal ultrasound scan shows a cirrhotic liver with associated ascites, but no evidence of hepatic or portal vein thrombosis.

**Learning Issues**

Polymorphonuclear count of >250/mm³ on ascitic fluid analysis is diagnostic of spontaneous bacterial peritonitis

PACT module on Coma and altered consciousness
PACT module on Clinical imaging
PACT module on Abdomen in acute/critical care medicine

Two days later the patient’s noradrenaline requirement has reduced to 0.1 mcg/kg/min, the urine output is >0.5 mL/kg/hour, and peripheral perfusion has improved. Lactate, although reduced, remains elevated at 4 mmol/L.

Q. What is your interpretation of the lactaemia - despite the resolution of the circulatory shock?

A. The clearance of lactate may be slow due to hepatic dysfunction.

**NOTE** Always interpret values in clinical context. A patient who has warm peripheries with good urine output and minimal vasopressor requirement does not have significant haemodynamic shock.

**Learning Issues**

Clinical assessment
Causes of hyperlactaemia

On day three the patient’s oxygen requirement increases to FiO₂ 0.8. CXR now shows diffuse bilateral pulmonary infiltrates. PiCCO readings are:

- PPV 5%
- GEDI 870 mL/m²
- EVLWI 15 mL/kg
- PVPI 4

Q. You suspect evolving ARDS and you note that the patient has positive fluid balance. What do you do now?

A. Active diuresis is indicated.

Q. How would you ventilate the patient?

A. You utilise lung protective ventilation by maintaining the tidal volume at 6 mL/kg, and titrate up the PEEP.
Recognising that the patient is now on the flat part of the Frank-Starling curve will change your haemodynamic management. Offloading fluid and aiming for a negative balance becomes a priority, especially if ARDS has developed.

The patient spends a further 18 days in the ICU, with a course complicated by ventilator-associated pneumonia and delirium. Tracheostomy is performed as he is slow to wean from mechanical ventilation. He is decannulated on day 21 of hospital admission and discharged home a week later.

On reflection, these cases demonstrate how a systematic approach to haemodynamic monitoring, together with the early utilisation of measured information, can be applied whatever the clinical scenario. The concept of maintaining adequate global and tissue perfusion is central to both monitoring and management. Frequent reassessment of the indices of organ and tissue perfusion and keeping an open mind are mandatory. Note the importance of the diagnostic information concurrently obtained in shaping therapy. Ongoing critical appraisal of the information is mandatory for optimum management and to minimise errors.