Non-invasive & Invasive Monitoring in ITU

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Associated clinical guidelines/protocols:
- Arterial lines: insertion & management
- CVL

Fundamental Knowledge:
List of topics relevant to PIC that will have been covered in membership examinations.
They will not be repeated here.
- The role of clinical assessment in monitoring;
- Physical principles underlying the use of monitoring devices;
- Indications for and contraindications to the use of monitoring devices;
- Interpretation of information from monitoring devices, and
- Principles of pressure transducers

Information for Year 1 ITU Training (basic):

Year 1 ITU curriculum
- General Principles
- Non invasive monitoring:
  Respiration – Pulse oximetry, ETCO2
  Cardiovascular - Pulse and blood pressure, cardiac output monitoring
  Temperature
  Nervous system: Neuromuscular junction, sedation
- Invasive Pressure Monitoring – general principles.
- Arterial catheterisation Components of the arterial waveform & the relationship to the ECG. Effects of haemodynamic disorders on the waveform, effect of respiration. Principles of zeroing, calibration, damping, catheter whip. Sites to be avoided.
- Complications
- Central venous catheterization: components of central venous waveform, effects of respiration and positive pressure ventilation. Risks and complications.
- Intracranial pressure monitoring – see CNS anatomy & pathophysiology module.
Curriculum Notes for Year 1:

General Principles:
- *Despite the wonders of modern technology, the value of clinical observations cannot be overemphasized;*
- *Monitors are unlikely to be of value unless the user can recognize, interpret and act appropriately on what is displayed.*

Increasingly sophisticated instruments are being used for monitoring and guiding patient care but the onus is on the observer to be aware of the limitations of measurements and the causes of error.

Confounding errors in clinical measurements are readings that are not true reflections of the underlying signal. They may be caused by instrumental, sampling and patient factors.

The measuring system must be accurate and precise to produce reliable clinical measurements. **Accuracy** is the difference between the measurement and a ‘gold standard’ measurement of the underlying signal. **Precision** is the reproducibility of repeated measurements of the same biological signal. A repeated consistent measurement may be precise but inaccurate, for example an erroneous arterial pressure in an un-zeroed system.

The bottom line: *if the data from the monitors does not accord with the patient’s clinical status, check the systems rigorously before implementing treatment changes.*

**NON INVASIVE MONITORING**

**RESPIRATION:**
- a) *Blood gas measurement;*
- b) *Pulse oximetry;*
- c) *Capnometry*

**a) Blood gas measurement**
- Traditional blood gas analysers report many results, but the only parameters directly measured are the partial pressures of oxygen (pO₂) & carbon dioxide (pCO₂) & blood pH. The haemoglobin saturation (HbO₂%) is calculated from the pO₂ using the oxygen-dissociation curve and assumes a normal P50 and that there are no abnormal forms of haemoglobin.
- Some blood gas analysers incorporate a co/oximeter that directly measures the various forms of haemoglobin including oxyhaemoglobin, total haemoglobin, carboxyhaemoglobin and methaemoglobin.
- The actual bicarbonate, standard bicarbonate, and base excess are calculated from the pH and pCO₂ using the Siggard-Anderson nomogram derived from a series of *in vitro* experiments relating pH, pCO₂ and bicarbonate.

**Practical precautions:**
- A heparinised, freshly drawn, bubble-free, arterial blood sample is required unless immediate point-of-care analysis is used.
- Heparin is acidic and if too much is present, the measured pCO₂ and calculated bicarbonate are spuriously reduced.
- Delay in measurement allows continued metabolism by the erythrocytes and reduces pH and pO₂ and increases pCO₂.
- Keeping the specimen on ice slows metabolism allowing accurate measurement to be delayed for up to 1 hour.
- Air bubbles introduce error and cause a fall in pCO₂ and an increase in pO₂.

**Temperature considerations:**
- Solubility of all gases in blood ↑ with a ↓ in temperature (including CO₂ and O₂)
• Patient hypothermia causes the pO$_2$ and pCO$_2$ to fall and the pH to rise.
• Analysis of a sample taken from a hypothermic patient usually occurs at 37°C resulting in artificially high pO$_2$ & pCO$_2$.
• Results can be corrected to the patient’s temperature, but in practice is usually unnecessary.

b) **Pulse oximetry:**

**Principles of operation – the simple version:**

- The absorption spectra of oxygenated and reduced haemoglobin differ
- 2 compounds with different absorption spectra together in solution: the ratio of their concentrations is determined from the ratio of light absorbed at 2 different wavelengths.
- A probe is placed on the finger, toe, ear lobe or nose.
- 2 light-emitting diodes produce beams at red & infrared frequencies (660 nm & 940 nm).
- The diodes flash + 30 /second.
- The diodes emit in sequence, with a pause with both diodes off ~ allowing compensation for ambient light.
- A photo detector on the other side of the digit (or earlobe, etc) analyses the change in light absorption that has occurred at the two wavelengths and determines the ratio of oxygenated:reduced haemoglobin – i.e. haemoglobin oxygen saturation.
- In living tissue, light is also absorbed by the tissues and the haemoglobin in venous and capillary blood
- The arterial saturation is measured by analysing the pulsatile changes in light absorption and ignoring/deducting the non-pulsatile (venous & tissues) component of the signal.
- **Clearly, pulsatile blood flow into the digit is necessary for proper analysis.**
- The measurements are plotted against a standard calibration curve, determined by direct measurements of the arterial oxygen saturation in normal resting healthy volunteers; accuracy is ± 2% between 85 and 100%
- Below 70% readings are extrapolated: - it was deemed not ethical to induce such hypoxia in the volunteers!

**NB**

- Anaemia does not affect readings unless haematocrit is below 10%
- Fetal haemoglobin has the same absorption spectra, so is accurate in neonates
- Hyperbilirubinaemia does not affect accuracy;
- Sickle haemoglobin does not affect readings (but HbO$_2$ curve is shifted);
- Skin colour does not influence accuracy.
- Pulse oximetry derived O2 saturations are denoted as SpO$_2$, rather than SaO$_2$

**Important Limitations:**

- Readings are averaged every 10-20 seconds; so they cannot detect acute desaturation.
- Finger probes have response times of 30-60 secs; earlobe probe of about 15secs.
- Pulse oximeters estimate arterial haemoglobin O2 saturation (SaO$_2$) and not arterial oxygen tension (PaO$_2$). Because of the shape of the oxygen-dissociation curve, large changes in PaO$_2$ may occur at the extremes of the curve with minimal change in SaO$_2$.
- Pulse oximetry is a global measure of functional oxygen saturation; it reveals nothing about adequacy of ventilation (e.g. PaCO$_2$), or oxygen content (e.g. a grossly anaemic patient with deficient oxygen content & delivery to the tissues with normal saturations).
- Poor peripheral pulsatile blood flow, e.g. vasoconstriction, low cardiac output, or arrhythmias, results in an inadequate signal for analysis.
- Venous congestion may produce venous pulsations which can cause erroneously low readings.
- Pulse oximetry is accurate above 90% saturation, but much less so below 70%.
- They are highly inaccurate below 50%.

**Errors:**

The most common causes are signal artefacts, which produce a poor signal-to-noise ratio.

- Patient movement is commonest source of error
• Excess ambient light, fluorescent lighting, infrared heaters and diathermy interfere with accuracy as the light detectors cannot differentiate the red light LED wavelength from other sources of light.

• Poor perfusion with poor pulsatile signal introduces error.

Other sources of error:
• Carboxyhaemoglobin has a similar absorbance to oxyhaemoglobin and gives a falsely high reading for SpO\textsubscript{2}. (SpO\textsubscript{2} reads 95% at 50% carboxyHb content)
• Methaemoglobin has an absorption that is similar at 660 and 940 nm, and produces a SpO\textsubscript{2} around 85%.
• IV dyes methylene blue & indocyanine green lead to falsely low SpO\textsubscript{2}.
• Nail varnish (especially blue colours) & dirt decrease SpO\textsubscript{2} reading.

NB
**Haemoximeters** (co-oximeters) work on spectrometric principles and directly measure the ratio of the oxygenated haemoglobin to the total haemoglobin in a sample of blood (SaO\textsubscript{2}). They will also directly measure other haemoglobin species, eg methaemoglobin or carboxyhaemoglobin.

**c) Capnometry**
Capnometry is the measurement of end tidal FCO\textsubscript{2} at the airway opening. End-tidal gas represents alveolar gas and in normal lungs the EtCO\textsubscript{2} tension is 0.5–0.8 kPa less than the arterial CO\textsubscript{2} tension, and so a non-invasive estimate of arterial CO\textsubscript{2}

Capnography is the graphic display of measured FCO\textsubscript{2} against time or volume

EtCO\textsubscript{2} analysers must have a rapid response time. Most devices measure CO\textsubscript{2} tension from the absorption of infrared light, though other techniques such as mass spectrometry can be used.

**Sidestream sampling** - continual aspiration of a gas sample of from the respiratory circuit which is fed through the analyser. The gas is returned to the respiratory circuit or scavenged. Used frequently during anaesthesia ~ convenience of a lightweight attachment to the airway. Errors occur if the sampling line becomes blocked (humified gas, water vapour, secretions).

**Mainstream sampling** – the analyser head is attached directly to the airway and the gas is analysed within the respiratory circuit through a clear window or cuvette. This adds dead space to the circuits.

More common capnograph: EtCO\textsubscript{2} vs time. Note these have inspiratory and expiratory phases.

Inspiration: CO\textsubscript{2} tension should be negligible, unless there is rebreathing.
Expiration: dead space gas (no CO2) is exhaled first, followed by alveolar gas & a rapid rise in CO2 which reaches a clear plateau in normal lungs and is termed the end-tidal CO2 tension.

Fig. 22. The 3 phases of a volumetric capnogram. Phase I: The volume of carbon-dioxide-free gas. Phase II: Transition from carbon-dioxide-free gas with the volume of early-empting alveoli. Phase III: Alveolar plateau with a positive slope that indicates a slowly rising volume of carbon dioxide. (Adapted from Reference 49, with permission.)


EtCO2 vs expired tidal volume.

Disease of the lung can result in significant non-homogeneity of ventilation within the lungs and no clear plateau is discernible so an accurate end-tidal CO2 cannot be measured. The transition phase is prolonged. (see below). This occurs in obstructive airway disease and asthma. In addition, if tidal volumes are very small there is inadequate distinction between dead space and alveolar gas resulting in an indistinct expiratory plateau

Fig. 3. Capnogram from a patient with chronic respiratory disease shows (A) transition phase is longer than normal (shaded area). B: A large tidal volume with a prolonged expiratory phase reflects PaO2.


The difference between end-tidal and arterial CO2 reflects the degree of V:Q matching. Normal (a-ET) CO2 is 2-5mmHg
(a-ET) CO2 can be used as a measure os V:Q mismatch or alveolar dead space. If the is significant mismatch, the end-tidal CO2 may markedly underestimate arterial CO2 tension.

- (a-ET) Co2 is ↑ with age, asthma, emphysema
- A reduction in lung perfusion results in an end-tidal CO2 tension lower than the arterial CO2 tension. This occurs in pulmonary emboli, low cardiac output states, hypotension and following the application of high levels of PEEP.
- A sudden drop in ET CO2 may reflect a sudden ↓ in pulmonary perfusion: i.e., ↓ CO or pulmonary embolism (thrombus, gas, fat).
- Monitoring end-tidal CO2 is useful during cardiopulmonary resuscitation for assessing adequacy of cardiac output.
ETCO2 can occasionally be > PaCO2 in circumstances of low FRC and high PaCO2 e.g. infants and pregnant women.

Increased ETCO2:
1. Alveolar hypoventilation
   a. ↓ respiratory rate
   b. ↓ tidal volume
   c. ↑ mechanical dead space
   d. Partial airway obstruction
2. Increased CO production
   a. Fever
   b. Catabolism
   c. Bicarbonate
3. ↑ pulmonary perfusion
4. Equipment malfunction

Decreased ETCO2
1. Alveolar hyperventilation
   a. ↑ respiratory rate, tidal volume
2. Absent alveolar ventilation
   a. Apnoea
   b. Complete obstruction of airway
3. ↓ CO2 production
   a. hypothermia
4. ↑ V:Q mismatch
   a. Pulmonary embolism
   b. Hypotension
   c. Hypovolaemia
   d. Cardiac arrest
5. Equipment malfunction/sampling error
   a. Inadequate tidal volume
   b. Sampling tube blocked
   c. Circuit disconnection

Errors:
Modern CO₂ analysers are based on the absorption of infrared light by CO₂ in a gas sample based on the Beer–Lambert law. There are many sources of error. There must be no leaks in the breathing system, the non/rebreathing valve should be functioning perfectly and the inspired gas well mixed and of constant composition.

Errors related to the machine;
- overlap of absorption wavebands of different gases; nitrous oxide (N2O) has an identical molecular weight to CO₂, so absorbs some of the infrared energy within the CO₂ bandwidth, and the CO₂ measurement is falsely high. (Only important in PICU if Entonox is being used)
• Modern infrared analysers provide a simultaneous breath-by-breath analysis of CO2, O2, N2O and volatile agents. Microprocessor technology can automatically identify and correct for the presence of other gases. Exhaled alcohol can confound vapour concentration in these machines.

Response time – a capnograph needs a rapid response time to reach the true maximum values at normal breathing frequencies. A slow response time will show prolonged phase II with a steeper phase III (similar to asthma), ↓ in peak end-tidal CO2, ↑ baseline

Water vapour – a slow response time is caused by long tubing and blockage in the sampling line. Liquids and particulate matter entering the measuring chamber cause erroneous readings of CO2 owing to their high infrared absorbance.

Errors related to sampling:
• patient size, respiratory rate and site of sampling may contribute to sampling errors.
• the optimal sampling site is at the top of the tracheal tube.
• Dilution of the end-tidal sample by high fresh gas flow; a right-angled connector should be used to prevent this.
• High flow rates on sampling in expiration can introduce error at low tidal volumes and fast respiratory rates (e.g. neonates & infants).
• The response time of the analyser should be less than the respiratory cycle time to achieve predictable CO2 values and waveform.
• In smaller children and neonates a sine wave type of capnogram can occur during spontaneous ventilation where there is no clear alveolar plateau or with controlled ventilation in neonates.

Transcutaneous gas analysis:
These are rarely used at the present time.
Transcutaneous oxygen analysers (PtcO2) placement of a Clark-type sensor on the skin, heated to 43°C to produce localized hyperaemia to maximize local capillary blood flow. Tissue oxygen diffuses across the epidermis to sensor and measured, with correlation of 0.90 with arterial blood gas analysis. In contrast to oximetry, oxygen tension rather than saturation is measured. A 15 minute equilibration and calibration time is needed.
Sensor dislodgement, poor calibration, skin oedema, low cardiac output, hypothermia, and severe acidosis results in inaccuracy. There is risk of skin damage from the sensor.
Transcutaneous carbon dioxide analysers (PtcCO2) use a pH sensitive glass electrode that is placed on the skin occlusively, and heated to 44°C. CO2 equilibrates across the epidermis effecting a change at the pH electrode that is proportional to the PaCO2. Disadvantages arise from the slow equilibration time and a need for frequent sensor changes (every few hours) to limit skin damage, but burns still occur, especially where there is poor tissue perfusion.

Non- Invasive cardiac monitors:
Heart rate is traditionally obtained from the ECG, pulse rate from pulse oximeter or invasive arterial monitor.

ECG Trace
Potentials from the heart are transmitted through the tissues, detected by electrodes, which produce an ECG recording. Silver and silver chloride forms a stable electrode combination. They are separated from the skin by a foam pad soaked in conducting gel. The ECG signal is boosted by an amplifier, which also filters out noise. The amplified ECG signal is then displayed on an oscilloscope.

Monitoring mode
Frequency response of 0.5-40 Hz. All ECG monitors use filters to narrow the bandwidth to reduce environmental artefacts. High-frequency filters reduce distortions from muscle movement, mains current and electromagnetic interference from other equipment. Low-frequency filters provide a stable baseline by ↓ respiratory and body movement artefacts.

Diagnostic mode
This mode monitors the ST-segment and there is a greater need for filtering of the signal

Electrode configurations
Most monitors display a single ECG trace using three electrodes: right arm, left arm and indifferent on left leg (or more usually left chest). Leads I, II & III can be displayed, but Lead II
is best for detecting arrhythmias. In some situations, e.g. LV ischaemia, may be better to use CM5 (Right arm electrode on manubrium, left arm electrode at V5 and indifferent lead on left shoulder); this lead detects approx 90% of ST-segment changes.

**Sources of error**
The ECG measures potentials of 0.5–2 mV at the skin surface. Noise and interference that obscure these tiny signals are the main cause of a poor ECG trace.

*Noise originating from the patient-electrode interface* – high skin impedance is the most common reason for a poor ECG signal. Reduced by de-greasing the skin with alcohol. Modern disposable electrodes are made of silver coated with silver chloride with stable impedances reducing slow drift. Gel-impregnated foam pads decrease movement artefact and lower skin impedance.

*Noise originating from the patient* – high frequency electromagnetic activity caused by shivering or movement is filtered by low-pass filters. Interference caused by shivering may be improved by placing the electrodes over bony prominences.

*Incorrect electrode placement*
The most obvious error is incorrect electrode placement relative to the heart because QRS complexes vary with position of the electrodes.

*Noise originating from the environment* – i.e. electrical interference. This is the distortion of a biological signal by capacitance or inductance effects. Any electrical device, powered by AC, can act as one plate of a capacitor and the patient acts as the other plate (e.g. warming mattress). This may cause a current with AC frequency to flow in the ECG leads, seen as 50Hz interference in all leads. Interference may also result from high frequency diathermy if surgical procedure being undertaken in ICU. Shielding of cables and leads, differential amplifiers and filters help to reduce such interference. The shielding consists of woven material, which is earthed. Interference currents are induced in the metal screen and not in the monitoring leads. The screening layer may often be covered by a second layer of insulation.

**NON-INVASIVE BLOOD PRESSURE MEASUREMENT**
There are several techniques of non-invasive blood pressure measurement (NIBP), all of which function by occluding the pulse in a limb with a proximal cuff, then detecting its return distally on lowering the occluding pressure.

Detection methods include: * = manual methods
- Palpation*
- Auscultation*
- Oscillotonometry*
- Oscillometry (e.g. Dinamap)
- Plethysmography (the Finapres)

Indirect methods of blood pressure measurement provide intermittent readings that overestimate at low pressures and underestimate at high pressures. Readings vary according to the site of measurement, owing to hydrostatic effects and increased arterial pulse wave velocities in the peripheries.

**Manual methods** –
Hardly ever used now in the intensive care environment.

Errors in the auscultatory method occur because of deficiencies in the transmission of sound owing to overlong or loose stethoscope tubing. Korotkoff sounds are difficult to hear in hypotension and arteriosclerosis, resulting in underestimation of blood pressure. Shivering patients require high occluding pressure, producing pseudohypertension.

Further measurement errors result from confusion over whether phase IV (muffled tone) or V (no tone) is to be taken as the diastolic point. Phase V is closer to directly measured diastolic pressure but may be absent in high output conditions (e.g. aortic regurgitation, warm shock). Oscillotonometry involves detection of the returning pressure with a second cuff, which causes a pointer to pulsate.

Common sources of error are wrong cuff size, zero and calibration errors in aneroid manometers, and leaks from the pneumatic system that prevent a slow and controlled deflation of the cuff.
Automatic methods –

- Oscillometry uses a single cuff both to compress the artery and to detect pulsations.
- A microprocessor controls the sequence of inflation and deflation of the cuff.
- Cuff is inflated to a pressure above the previous systolic pressure & then deflated incrementally.
- A fast rate of inflation and a slow cuff deflation avoids venous congestion and allows time to detect arterial pulsation.
- A transducer senses the pressure changes, which are processed by the microprocessor. This has an accuracy of +/- 2%.
- As mean arterial pressure is reached, these pulsations reach maximum amplitude
- The original Dinamap only measured mean arterial pressure, hence the acronym: Device for Indirect Non-invasive Automatic Mean Arterial Pressure (DINAMAP)

More sophisticated algorithms allow systolic and diastolic pressure to be measured. The systolic pressure corresponds to the onset of rapidly increasing oscillations. Diastolic pressure corresponds to the onset of rapidly decreasing oscillations. It is also calculated from the systolic and MAP:

\[ MAP = DBP + \frac{(SBP - DBP)}{2} \]

- Systolic pressure: bias towards overestimation at low pressures & underestimation at high pressures.
- The principle relies on a regular cardiac cycle; inconsistent readings may occur in patients with rhythm disturbances.
- Over-frequent readings may cause impeded blood flow producing inaccurate results.
- Newer devices deflate the cuff continuously rather than in steps, thus achieving a shorter measurement cycle.
- The apparatus becomes inaccurate at pressures below 50 mmHg
- The cuff should be at least two-thirds of the upper arm.
- The width of the cuff's bladder should be 40% of the mid-circumference of the limb. The middle of the cuff should overlay the brachial artery.
- The cuff can also be placed on the thigh or calf.

Sources of error

- If the cuff is too small, the blood pressure over-reads;
- If too large then the blood pressure under-reads
- Systolic pressure over-reads at low pressures (<60 mmHg) & under-reads at high systolic pressures;
- Arrhythmias such as atrial fibrillation affect accuracy.

Complications

- Frequent, repeated inflations can cause ulnar nerve palsy and petechial haemorrhage of the skin underlying the cuff.

Temperature:

Core temperature: temperature of blood perfusing vital organs.

Small gradients exist between different parts of the body, so the extent a measured value reflects core temperature depends on the site chosen.

Skin temperature is a very poor indicator of core temperature, as influenced heavily by peripheral perfusion. The temp ‘gap’ between skin & core is used as an indicator of CO or peripeeral perfusion.

The following sites may be used for core temperature:

- Tympanic membrane: is a good indicator of core & brain temperature; probably ‘gold standard’; usage limited by concerns about damage to tympanic membrane
- Oesophagus; accurate in the lower 25% of the oesophagus; good estimate of cardiac temperature.
- Nasopharynx; fairly accurate measurement of core temp, less accurate than oesophageal probe; position just behind the soft palate. Incorrect placement (ie too far) inaccurate results due to the cooling effects of inspired gasses.
Rectum  Rectal temperature is influenced by cooling effect of blood returning from lower limbs and heat generated by the thermogenic gut flora. The insulating effect of faeces results in rectal temperature being 0.5-1.0°C higher than core temperature and response time is slow.

Bladder  Bladder temperature is determined primarily by urine flow; high flow rates are necessary for temperature to reflect core temperature. Low urinary flow makes bladder temperature difficult to interpret in relation to true core temperature.

Blood  measured directly using a thermistor attached to a PA catheter; but very invasive.

Measurement techniques:
Non-electrical techniques
The traditional mercury thermometer has been withdrawn from the NHS because of concerns about mercury poisoning and cross-infection; as an alternative there are:

- Chemical thermometers;
- Infrared thermometers;

Chemical thermometers use heat-sensitive liquid crystals composed of chemicals such as cholesterol esters.

- Example - the Tempadot, a plastic strip impregnated with heat-sensitive chemical dots, which, when exposed to an increase in temperature, melt, resulting in a change of colour.
- faster reaction time than mercury, 1 min for an oral, 3 min for axillary reading.
- disposable and reduce the risk of cross-infection.
- scales are accurate only to 0.5 °C. Used for monitoring skin temperature

Infrared thermometers:  the frequency of infrared energy emitted by an object changes with temperature and can be measured.

- Infrared tympanic thermometers (ITTs) measure the frequency of infrared light emitted by the tympanic membrane.
- the external auditory canal must be free from wax prior to use.
- ITTs can be used as continuous temperature monitoring:

Electrical techniques
There are three principal electric techniques for measuring temperature:

i) Resistance thermometer
ii) Thermistor
iii) Thermocouple

Most ICU temperature probes are thermocouple based.

Non Invasive Cardiac Output monitoring.

a) Doppler (echo, oesophageal, suprasternal)
Simplistically, using the Doppler principle, blood velocity in a vessel can be calculated from the frequency shift of the reflected ultrasound wave. This is most commonly measured in the aorta. By integrating the velocity with the cross-sectional area of the aorta flow can be estimated, and hence cardiac output.

Basic Principles

**BLOODflowVOLUME**
The basic volume calculation is:  \[ \text{Volume} = \text{Area} \times \text{Height} \]
The amount of blood travelling across the aortic or pulmonary valve in one beat can be measured as a volume.

**Cross Sectional Area**
The area of the valve or outflow tract can be used in the volume equation.
If the diameter of the valve or outflow tract (OT) is known, the area can be calculated.

\[ \text{Diameter of the outflow tract (OTD) is 1.9cm} \]
\[ \text{OT area} = \pi \times r^2 \]
\[ \text{OT area} = 2.8 \text{ cm}^2 \]
**Stroke Distance - Velocity Time Integral**

When the volume equation is applied physiologically, the distance a column of blood travels in one beat is called the stroke distance. The stroke distance is measured by Doppler as the velocity time integral (v.t.i.).

v.t.i is a method of calculating the distance blood has travelled, allowing for the velocity and time that the blood has been moving.

\[
\text{Stroke Volume} = \text{OT Area} \times \text{Stroke Distance (v.t.i)}
\]

If the blood travels 20 cm with each beat, then:

\[
\text{Stroke Volume} = \text{OT Area} \times \text{v.t.i} \\
= 2.8 \text{ cm}^2 \times 20 \text{ cm} \\
= 56 \text{ cm}^3 \\
\text{Stroke Volume} = 56 \text{ ml}
\]

**OUTFLOW tract DIAMETER**

The aortic or pulmonary outflow tract diameter is used to calculate area for stroke volume (SV) and cardiac output (CO).

An anthropometric algorithm can be used to calculate flow volume. In adults and children, the OTD has been shown to correlate linearly with height. Usually the OTD is measured directly from a two-dimensional echocardiogram. A two-dimensional echocardiogram independent method for calculating flow volumes from Doppler spectral flow can also be used. This is a simpler method which is more accurate.

\[
SV = \text{CSA} \times \text{v.t.i}
\]

Right sided \( SV \) = Left sided \( SV \)

So:

\[
PV (\text{CSA} \times \text{v.t.i}) = AV (\text{CSA} \times \text{v.t.i})
\]

In children, the correlation is constant for both weight and height. As weight is a more commonly used measurement, it is used in the algorithm for neonates under 50 cm in height.²

**DOPPLER flow PROFILES**

The characteristic systolic flow profile is triangular shaped and provides information on:

- **VELOCITY**: the speed the red blood cells are travelling at a given time
- **DIRECTION OF FLOW**: toward or away from the transducer
- **TIMING**: systolic ajection time and diastole
- **INTENSITY**: the greater the intensity, the greater the number of red blood cells moving at that velocity

The non-invasive monitor is able to give the following haemodynamic measurements from the derivations above, including HR, BP and CVP.

**HAEMODYNAMIC measurements**

**Paediatric Ranges aged 2.5 to 16 years**

<table>
<thead>
<tr>
<th>CO - Cardiac Output</th>
<th>CO - SV x HR</th>
<th>Paeds 3.5 - 7.0 l/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cl - Cardiac Index</td>
<td>Cl = CO / BSA</td>
<td>Paeds 3.4 - 5.0 l/min/m²</td>
</tr>
<tr>
<td>SV - Stroke Volume</td>
<td>SV - v̇̇x CSA</td>
<td>Paeds 40 - 95 ml</td>
</tr>
<tr>
<td>SVI - Stroke Volume Index</td>
<td>SVI = SV / BSA</td>
<td>Paeds 40 - 60 ml/m²</td>
</tr>
<tr>
<td>SVV - Stroke Volume Variation</td>
<td>SVV = (SV_max - SV_min) x 100 / (SV_max + SV_min)</td>
<td>Paeds 20 - 60 ml/m²</td>
</tr>
<tr>
<td>SVR - Systemic Vascular Resistance</td>
<td>SVR = B0x(MAP-CVP)/CO</td>
<td>Paeds 900 - 1700 dynes/sec/cm²</td>
</tr>
<tr>
<td>SVRI - Systemic Vascular Resistance Index</td>
<td>SVRI = SVR / BSA</td>
<td>Paeds 1200-2000 dynes/sec/cm²/m²</td>
</tr>
<tr>
<td>VpK - Peak Velocity</td>
<td>Applies to valve being measured</td>
<td></td>
</tr>
<tr>
<td>Aortic Peak Velocity (ESN)</td>
<td>Paeds 1.2 - 1.6 m/s</td>
<td></td>
</tr>
<tr>
<td>Pulmonary Peak Velocity (LSU)</td>
<td>Paeds 0.7 - 1.1 m/s</td>
<td></td>
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</tbody>
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<table>
<thead>
<tr>
<th>HR - Heart Rate</th>
<th>Infants - 10 years</th>
<th>10 years - Adults</th>
<th>100 - 160 bpm</th>
</tr>
</thead>
<tbody>
<tr>
<td>FT - Flow Time</td>
<td>FT = Tc</td>
<td>Paeds 200 - 300 ms</td>
<td></td>
</tr>
<tr>
<td>LT % - Ejection Time Percentage</td>
<td>LT(1 - Cycle Duration)x100</td>
<td>Paeds 37 - 51 %</td>
<td></td>
</tr>
<tr>
<td>vti - Velocity Time Integral</td>
<td>vti = ∫(v(t) dt)</td>
<td>Paeds 23 - 33 cm</td>
<td></td>
</tr>
<tr>
<td>MD - Minute Distance</td>
<td>MD = vti x HR</td>
<td>Paeds 10 - 12 years</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>15 mmHg</td>
<td>AV 18 - 27 mmHg</td>
</tr>
</tbody>
</table>

References:

- BE Smith, AD Parakkal RAPID EVALUATION OF HAEMODYNAMICS IN THE CRITICALLY ILL PATIENT BY CONTINUOUS WAVE DOPPLER ULTRASOUND MEASUREMENT OF AORTIC MINUTE DISTANCE. Department of Anaesthetics and Critical Care, Broken Hill Base Hospital, NSW, Australia.
  1. The University of Queensland, Brisbane, Australia.

b) Transthoracic electric bioimpedance
This technique is based on the principle that electrical impedance across the thorax changes (by about 0.5%) with the cyclical change in blood volume that occurs with each heartbeat, ie the rate of change of impedance is a reflection of cardiac output. Voltage sensing and current transmitting electrodes are placed on either side of the thorax, and although the method has been reported to give accurate results in normal subjects, it is not reliable in critically ill patients. It has a low signal to noise ratio, and is probably only useful as a trend monitor, at best.

Other monitoring techniques:
Near infrared spectroscopy (NIRS) uses light in the near infrared range to assess changes in hemoglobin, blood volume, and tissue oxygen availability within the monitored tissue. NIRS uses the difference in light absorption between different components of the hemoglobin to determine oxygenation of the blood. At the cellular level through monitoring tissue oxygen availability. By monitoring transfer of electrons and the enzyme state, it provides valuable information on tissue oxygenation at the cellular level. Therefore it can be used to identify conditions of decreased tissue oxygenation.

Most work to date has been on cerebral NIRS, providing a continuous, direct, and noninvasive monitor of cerebral oxygenation and cerebral blood volume (CBV). NIRS may become the preferred method to monitor brain oxygenation and cerebral hemodynamics. It is safe, non-invasive and continuous, gives real-time information, and can be used at the bedside. It provides information on brain oxygenation at the tissue level. Concurrent with the assessment of oxygenation, it detects changes in brain blood volume, which in turn reflects alterations in brain blood flow and cerebral venous return.

Invasive pressure measurement:
General principles;
- A transducer is a device that converts one form of energy to another.
- In biomedical applications, transducers convert physiological signals to electrical signals, which are processed to provide observable information.
- In pressure monitoring, piezoelectric transducers are used which rely on certain materials to develop a voltage difference across them when a pressure is applied or the material is distorted i.e. they often rely on displacement of a diaphragm.
- The transducer is connected to an amplifier and oscilloscope, demonstrating the pressure waveform and displaying the measured pressure.
- In medicine, there is usually ‘hydraulic coupling’ of the site being measured to the transducer – in simple terms, a fluid-filled catheter.
- We must control damping & resonance (= under-damping) in the fluid-filled catheter to faithfully reflect the pressure signal.
- Damping is caused by dissipation of stored energy. Excessive damping causes loss of detail in the waveform; lowering systolic pressure, elevating diastolic pressure. Mean arterial pressure is unaltered.
- Damping results from air bubbles, blood clots, cannula kinking, arterial spasm, or a soft diaphragm.
- A common reason for a damped pressure trace is soft extension tubing.
- Resonance occurs if the tubing or diaphragm is too stiff. Depending on the shape of the arterial pressure wave, this distortion can introduce a 20–40% overshoot error in systolic blood pressure readings, and under-reading of diastolic pressure; it is worse with tachycardia.
• In practice, it has been found that the optimal situation is when the system is 64 – 70% damped

So:
• The catheter must be reasonably stiff and straight, and the fluid must not contain air bubbles.
• The use of relatively wide-bore cannulae and minimizing stopcocks improves the frequency response of the system.

Testing for optimal damping:
The amount of damping in a system can be assessed by snapping the flush valve and observing the response.

Catheter whip:
• similar to an under-damped waveform.
• due to long, flexible pulmonary artery catheter fluttering in a high velocity bloodstream.
• also occurs with intracardiac catheters due to excessive mobility of the catheter tip caused by cardiac contraction.
• These artifacts are generally unavoidable, given the nature of the catheters.
• It is important therefore to be aware of the phenomenon and to recognize that displayed PA or intracardiac pressures may over-estimate systolic and under-estimate diastolic pressures.

**Zeroing:**
• It is vital to calibrate the system accurately to atmospheric pressure (0)
• Opening transducer to atmosphere at its 'three way' tap, leveled to reference point (level of the tricuspid valve ~‘about’ mid-thorax).
• Unexpected changes in displayed pressure: ensure the calibration is correct and neither the bed height or transducer position have been unwittingly altered.
• Finally, infusions should not be administered through the CVP lumen of the catheter simultaneously with measurement, as this will artifactualy raise the readings.

**Invasive Blood pressure measurement:**
Involves placing a cannula within the blood vessel (artery or large vein) or a cardiac chamber, connected to a pressure transducer via a column of (usually) heparinised saline.
• To maintain catheter and vessel patency, the system is kept continuously flushed with a pressurized system (200-300mmHg)
• Saline passes through a drip chamber, at set flow -depending on the manufacturer -usually 3-4 ml/hour.
• In infants, this flow rate is too high, so the system is flushed using a syringe pump at 1-2 ml/hour.

**Arterial blood pressure measurement:**
• Intra-arterial cannulation with electromechanical transduction provides continuous monitoring of arterial blood pressure with greater accuracy than non/invasive methods.
• Damping and calibration errors account for most inaccuracies.
• The arterial pressure wave narrows and increases in amplitude in peripheral vessels, so the systolic pressure is higher in the dorsalis pedis than in the radial artery.
• Peripheral hypothermia can lead to damped traces, and inaccurately low pressures.

An optimally damped and accurate blood pressure tracing will look like this:

![Blood Pressure Waveform Diagram](http://www.anaesthesiaw.com/default.aspx)

• The mean pressure (MAP) is the average pressure throughout the cardiac cycle. Because systole is shorter than diastole, MAP is slightly less than the value halfway between systolic and diastolic pressure;
• A low dicrotic notch is seen in hypovolaemic patients;
• Slope of diastolic decay indicates resistance to flow. Slow fall is seen in vasoconstriction;
• A steep decay is seen with rapid run-off eg a shunt, aortic regurgitation;
• The slope of the upstroke of the wave reflects myocardial contractility (dP/dt);
The stroke volume can be calculated by measuring the area from the beginning of the upstroke to the dicrotic notch. If this is multiplied by the heart rate, then cardiac output can be estimated.

**Indications for invasive arterial pressure monitoring:** in general:
- Patients with unstable haemodynamics
- Patients requiring potent vasoactive drugs
- Where there is anticipated major fluid balance changes or bleeding
- Where there is a need for frequent arterial blood gas analysis

**Precautions in siting arterial lines:**
- Asepsis – of course;
- Securing the cannula; - it is easy to kink the cannula at the skin as it is taped in;
- Avoid using cannulae with injection ports – accidental injection of drugs into an arterial line is indefensible;
- Be careful also with taps used for sampling; - **use highly visible labelling to indicate it is an arterial line**;
- Ensure good fixation; arterial lines should not ‘fall out’;
- Ensure all connections are well secured; - it is easy to lose a lot of blood quickly through a poor connection;

**Ideally, Allen’s test** should be performed prior to siting a radial line to ensure there is collateral ulnar flow (inadequate in 3% of patients).

**Allen’s test**
1) Elevate hand to drain hand of blood as far as possible;
2) Firm pressure against both the radial and ulnar arteries for 1/2 minutes;
3) Hand should blanche white
4) Releases ulnar compression only

- **Normal result** - Hand colour flushes within 5 to 7 seconds
- **Abnormal result** - Hand remains white until radial pressure released; = Inadequate collateral circulation

**Complications:**
- Ischaemia
- Local skin necrosis- needs regular assessment. Blanching of the surrounding skin on flushing the arterial line can indicate a risk of ischaemic damage to the skin;
- Infection
- Bleeding
- Injudicious line flushing in upper limb lines can result in significant flushing back up to cerebral arteries.
- Inadvertent drug administration into an arterial line can cause major tissue or even limb necrosis, particularly from drugs with a high pH such as thiopentone.

**CENTRAL VENOUS PRESSURE (CVP) MEASUREMENT**

**Indications**
- unstable haemodynamics; i.e. as an indicator of intravascular volume;
- surrogate marker of right ventricular function;
- significant fluid management issues;
- Venous access when other peripheral sites are unavailable;
- Placement of a large-bore venous catheter
- Medication administration, particularly inotropes;
- Parenteral nutrition.
- As a conduit for Swan-Ganz (PA) catheters, temporary cardiac pacemakers, hemodialysis catheters.

**Contraindications**
Few – and all are relative;
- e.g.: Infection over the insertion site
- Bleeding diathesis

**Sites**
- Internal jugular
- Femoral
- Subclavian
- External jugular
- Long line from antecubital vein
- Umbilical (in newborns)

**Positioning**
- For intrathoracic lines, it is important to check position before use. This is usually done with CXR. **Optimal position is for tip to be at or just above junction of SVC and atrium.**
- In 2001, DOH issued guidance that tips should not be in atrium, following 4 cases of death following cardiac perforation in infants.
- In practice, it is acceptable for tip to be in large central vein; thrombosis is more common in smaller veins.

**Typical CVP Trace**

![Typical CVP Trace](http://domain675291.sites.fasthosts.com/anae/hdmon.htm)

- **a wave** - reflects RA contraction (may be elevated in RV failure)
- **c wave** - represents tricuspid valve closure and bowing of valve into atrium with ventricular contraction
- **v wave** - passive filling RA during ventricular systole; (elevated in tricuspid regurgitation)
- **x slope** - atrial diastole
- **y slope** - atrial emptying

**Normal CVP pressures:** 3-11 cmH2O / 0-8 mmHg

As CVP pressures are normally low it is particularly important that zeroing is accurate. **Inaccuracies**
- Systemic venoconstriction elevates CVP
- Decreased right ventricular compliance
- Obstruction of great veins
- Tricuspid valve disease or cardiac structural abnormalities
- Mechanical ventilation - elevates CVP in Inspiration.
- Note that during spontaneous ventilation, CVP falls during inspiration.

**Complications related to insertion:**
- Pneumothorax (subclavian, occasionally internal jugular)
- Carotid artery puncture (internal jugular)
- *Subclavian artery puncture (subclavian, internal jugular)
- Haemothorax
- Air embolism
- Arrhythmias
- Haematomas
- Venous congestion (femoral vein)

(NB; take particular care with the child with a BT shunt; hitting the subclavian artery may severely compromise shunt flow with dire consequences; avoid subclavian approach if possible).

**Long-term presence of line:**
- Infection.
- Catheter tip embolus or thrombotic embolus.
- Hematoma formation.
- Major vein thrombosis
- Pneumothorax, Haemothorax.
- Chylothorax.
- Air embolism.
- Arrhythmias.
- Bleeding
- Vascular erosions;
- Perforation atrium/ventricle
- Pericardial effusion / tamponade

**Coated catheters.**
Heparin coating of catheters is now widely available. The coating improves bio-compatibility, reduces the risk venous thrombosis and the incidence of bacterial colonisation.

The catheters are contraindicated in patients with heparin-induced thrombocytopenia.

Chlorhexidine and silver sulfadiazine impregnated central venous catheters are also available. Impregnation is intended to reduce the risk of catheter-related sepsis. There is as yet inconclusive evidence as to the effectiveness of this approach.

**Other sources of information:**

**Websites.**
http://www.capnography.com/ - very good interactive site.

**References.**
Information for Year 2 ITU Training (advanced):

### Year 2 ITU curriculum

- **Neuromuscular junction monitoring:** single twitch, tetanic stimulation and train of four stimulation
- **Infection rates of central catheters.**
- **Depth of sedation:** Bi spectral index monitoring
- **EEG – see CNS anatomy and physiology module.**
- **Pulmonary Artery Catheterisation:** Interpretation of pressure waveforms obtained on positioning catheter. Measurement of pulmonary artery, wedge pressure and cardiac output: Fick principle, thermodilution. Contra-indications (LBBB, intracardiac shunts) Complications (dysrhythmia, perforation, rupture, infarction).

### Curriculum Notes for Year 2:

**Neuromuscular junction monitoring:**

Neuromuscular blockade should be monitored regularly in patients on continuous infusions of muscle relaxants > 24 hours, particularly in the presence of renal dysfunction.

All techniques for assessing neuromuscular blockade use a peripheral nerve stimulator (PNS) to stimulate a motor nerve electrically. The PNS generates a standard electrical pulse, which should be:

- supramaximal to ensure recruitment of all available muscle units
- a square wave of short duration (0.1–0.2 ms) with uniform amplitude (10–40 mA).

The muscle response can be assessed by:

- Visual and tactile methods
- Electromyography
- acceleromyography and
- mechanomyography.

**Visual observation and palpation** of the contracting muscle group are the easiest but least accurate methods of assessing neuromuscular block from PNS stimulation.

**Electromyography** uses electrodes to record the compound muscle potential stimulated by the PNS. Typically, the ulnar nerve is used and the electrodes are placed over the motor point of adductor pollicis. A drawback is that small movements of the hand can change the response by altering the electrode geometry.

**Acceleromyography** – acceleration of a distal digit is directly proportional to the force of muscle contraction & therefore inversely proportional to the degree of neuromuscular block. The transducer uses a piezoelectric crystal secured to the distal part of the digit measured and the PNS provides the electrical stimulus. Accurate and stable positioning of the digit is important for accurate results.

**Mechanomyography** uses a strain gauge to measure the tension generated in a muscle. A small weight is suspended from the muscle to maintain isometric contraction. The tension produced on PNS stimulation is converted into an electrical signal. Mechanomyography requires splinting of the hand and is generally used for research.

**Different modes of PNS stimulation**

Usually delivered over the median or ulnar nerve, looking for response in hand muscles, or lateral popliteal nerve in the lower leg with response in the foot

**Single twitch** – an electrical pulse is delivered, and the ratio of the evoked twitch compared with that before muscle relaxation gives a crude indication of neuromuscular blockade. When 75% of the acetylcholine (ACh) receptors on the postsynaptic membrane of the neuromuscular junction are occupied by a neuromuscular blocking agent (NMBA), twitch magnitude starts to decrease. When there is 100% drug occupation, no twitch is elicited.
Train of four (TOF) – 4 stimuli are given at a frequency of 2 Hz, potentially eliciting 4 twitches (T1–T4). The ratio T4:T1 indicates the degree of neuromuscular block. Non-depolarizing NMBAs produce a decrease in magnitude of the first twitch compared with a pre-relaxant stimulus, and a progressive reduction in magnitude of T1–T4. The number of elicited twitches indicates the degree of receptor occupancy. Disappearance of T4, T3, T2, T1 corresponds to 75%, 80%, 90% and 100% occupancy. With recovery of neuromuscular function the twitches appear in the reverse order. Accepted values for TOF count are:

- 1 twitch for tracheal intubation
- 1–2 twitches during established anaesthesia
- 3–4 twitches before reversal of neuromuscular blockade is attempted.

It is generally unnecessary to achieve 100% neuromuscular block in patients in ICU. 80-90% blockade, ie 2-3 twitches is normally satisfactory.

Double burst stimulation consists of 2 bursts of 3 stimuli at 50 Hz with each triple burst separated by 750 ms. These manifest visually as two separate stimuli (T1 and T2). The ratio is related to the TOF ratio and is easier for the operator to interpret reliably.

Tetanic stimulation at 50 Hz for 5 s produces detectable fade in muscle contraction, the extent of which is related to neuromuscular block. No fade indicates no neuromuscular block. In intense neuromuscular block, TOF stimulation elicits no twitches. Post-tetanic potentiation (PTP) uses tetanic stimulation for 5 s to mobilize presynaptic ACh. Subsequent 1 Hz twitch stimulation can overcome the high concentrations of NMBAs. The number of twitches generated (i.e. the post-tetanic count) reflects the degree of neuromuscular blockade. A twitch count of no more than 8–10 will generally mean an adequate degree of block.

Depolarizing NMBAs (ie suxamethonium) react differently to the PNS modes of stimulation. They produce equal but reduced twitches in response to single twitch and TOF stimulation (the T4:T1 ratio is 1), reduced but sustained contraction with tetanic stimulation, but do not demonstrate either tetanic fade or PTP.

Depth of sedation:
The electrical activity of the brain provides information on depth of anaesthesia / sedation. The EEG detects voltages of 1–500 µV. It comprises alpha, beta, theta and delta waves. With increasing depth of sedation there is a progressive increase in signal amplitude and a reduced frequency (burst suppression). The EEG is non-invasive and presents cortical electrical activity derived from summated excitatory and inhibitory postsynaptic activity that is paced by subthalamic nuclei.
Different drugs have varying effects on the EEG, and hypotension, hypoxia, metabolic encephalopathy and cerebral oedema can all depress EEG signal output.

**Bispectral index (BIS)** The BIS monitor displays a real-time EEG trace, acquired from a frontotemporal montage. The monitor generates a dimensionless number on a continuous scale of 0-100, with 100 representing normal cortical electrical activity and 0 indicating cortical electrical silence. The influence of pre-existing neuropathology on BIS values is unknown. As with any EEG signal, BIS is subject to interference and artefact, particularly from electromyographic (EMG) activity, which can artificially elevate the recorded BIS. The display also shows a signal quality index and an indicator of EMG interference.

BIS values discriminate between awake and asleep states but with considerable overlap of values. BIS records a state of the brain and not the effect of a particular drug. BIS gives a numerical value and the data are generated over 30 EEG recordings, with the average updated every 2–5 s. A low BIS value indicates hypnosis. BIS decreases during natural sleep, though not to the level produced by anaesthetic drugs.

**PULMONARY ARTERY PRESSURE (PAP)**

Pulmonary artery pressure can be measured via a line placed at the time of cardiac surgery, or by passing a flow-directed multi-lumen catheter from a central vein via the right atrium and right ventricle into the pulmonary artery. These latter catheters are better known as Swan-Ganz catheters.

Proximal lumen: 25 cm from tip, lies in right atrium, measures central venous pressure (CVP). Distal lumen: at tip of catheter, lies in a branch of the pulmonary artery, connected to a pressure transducer. Balloon lumen: permits introduction of 1.5 ml of air into the balloon at the distal tip. Thermistor lumen: bead situated 4 cm from the tip of the catheter and measures temperature.

They were very common a few years ago, particularly in adult intensive care practice, but are hardly used at the present time, as there was no observed improvement in outcome, and a relatively high complication rate from their use. This remains a contentious area.

Current usage is predominantly in adults and older children and the following is therefore provided primarily for basic understanding.
**Pulmonary artery catheters** can provide useful hemodynamic data, particularly in low cardiac output of unknown cause. E.g. CVP does not always reflect left-sided cardiac preload, (i.e. systemic filling pressure), particularly in the context of significant lung injury.

Passing a multilumen catheter into the pulmonary artery can provide information not only about the state of the right side of the heart, but also about the left-side by assessing **pulmonary artery wedge pressure (Ppw)**. Measured from the right side of the heart, reflects the left sided pressure because during diastole the pulmonary venous bed and left ventricle are in direct communication.

True mixed venous saturations can be obtained.

**PA catheter insertion:**
The catheter is passed (aseptic conditions) via a sheath inserted into a big central vein (often IJV). It is fed into the RA, then the RV and then floated into the pulmonary artery with the aid of a small balloon at its tip. Monitoring the pressure during advancement of the line will show where the catheter is during its advancement. The RV will (usually) have a pressure close to 0 during diastole. The pulmonary artery waveform has a systolic pressure wave and a diastolic trough. A dicrotic notch due to closure of the pulmonary valve may be seen on the terminal portion of the systolic pressure wave.

http://www.anaesthesiauk.com/

**NB**
Arrhythmias are a frequent complication of PA catheter insertion (and removal).

The **Ppw** is obtained by inflating the catheter balloon per manufacturers instructions (usually 1-1.5 ml of air). This allows the PA catheter tip to advance in the pulmonary artery until it obstructs the forward flow of blood. This situation creates a static column of fluid distal to the tip of the catheter. The pressure recorded at the catheter tip (Ppw) is equivalent to the pressure in the occluded pulmonary vein and equates to left atrial pressure.

<table>
<thead>
<tr>
<th><strong>Right Atrium</strong></th>
<th><strong>0-8 mmHg</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Right Ventricle</strong></td>
<td>15-25 mmHg systolic; 0-8 mmHg diastolic</td>
</tr>
<tr>
<td><strong>Pulmonary Artery</strong></td>
<td>15-25 mmHg systolic; 8-15 mmHg diastolic</td>
</tr>
<tr>
<td><strong>Pulmonary Capillary Wedge Pressure</strong> (PCWP or Ppw)</td>
<td>4-12 mmHg; (less than PA diastolic) (Except Mitral regurgitation giant v waves)</td>
</tr>
<tr>
<td><strong>Mixed venous saturations</strong></td>
<td>70-75%</td>
</tr>
</tbody>
</table>

Obtaining reliable and accurate data from pulmonary wedge pressure requires skill and experience. Interpretation is complex and beyond the scope of this text. Please see refs.
INVASIVE CARDIAC OUTPUT MEASUREMENT
Cardiac output is the amount of blood pumped to the peripheral circulation by the heart every minute. It reflects the whole circulatory bed, not just the heart, as multiple factors influence it.

\[ \text{CO} = \text{SV} \times \text{HR} \]

Invasive Cardiac Output measurement is not frequently used in children, in part because of technical and size restraints, and the risk of complications.

Invasive CO measurement is based on the Fick principle, (described in 1870).

\[ Q = \frac{M}{V - A} \]

Where;
- \( Q \) is the volume of blood flowing through an organ in a minute,
- \( M \) is the number of moles of a substance added to the blood by an organ in 1 minute,
- \( V \) and \( A \) are the venous and arterial concentrations of that substance.

- This principle can be used to measure the blood flow through any organ that adds substances to, or removes substances from, the blood.
- The heart does not do either of these, but the CO equals the pulmonary blood flow, and the lungs add oxygen to the blood and remove carbon dioxide from it.

So, the basic principle of CO measurement uses the oxygen consumption (the amount of oxygen taken up by the lungs in a minute), and the arterio-venous difference of oxygen across the lungs:

\[ \text{CO} = \frac{\text{VO}_2}{\text{C}_{\text{PV}}\text{O}_2 - \text{C}_{\text{PA}}\text{O}_2} \]

Where:
- \( \text{C}_{\text{PV}}\text{O}_2 \) is the oxygen content of pulmonary venous blood,
- \( \text{C}_{\text{PA}}\text{O}_2 \) is the oxygen content of pulmonary arterial blood,
- \( \text{VO}_2 \) is the oxygen consumption.

\( \text{NB: Blood O2 content} = \text{Hb} \times \text{Sa O2} \times 1.36 = \% \text{ ml O2 in 100 mL blood} \)

In the normal situation, pulmonary venous oxygen content will be the same as arterial oxygen content, and pulmonary venous oxygen saturation will be the same as mixed venous oxygen content:

\[ \text{CO} = \frac{\text{VO}_2}{\text{C}_a\text{O}_2 - \text{C}_v\text{O}_2} \]

Where:
- \( \text{C}_a\text{O}_2 \) is the oxygen content of arterial blood,
- \( \text{C}_v\text{O}_2 \) is the oxygen content of mixed venous blood;

Oxygen consumption is measured indirectly using lung oxygen uptake, arterial oxygen content from an arterial blood sample, and mixed venous oxygen content from a pulmonary artery sample from a pulmonary artery catheter.

\( \text{For most practical purposes, SVC or right atrial blood is used as} \ '\text{mixed venous blood}' \)

The technique can only be applied under steady state conditions but the accuracy and reproducibility are good. This is the gold standard for cardiac output measurement, and all other invasive methods are based on this principle.

Limitations
- The original method described by Fick is difficult to carry out.
- \( O2 \) consumption is derived by measuring expired gas volume over a known time & the difference in \( O2 \) concentration between this expired gas and inspired gas.
- Accurate collection of the gas is difficult unless the patient has an ETT.
- Analysis of the gas is straightforward if the inspired gas is air, but if it is oxygen-enriched air there are two problems,
  (a) addition of oxygen fluctuates & error due to non-constancy of the inspired \( O2 \) concentration
  (b) it is difficult to measure small changes in \( O2 \) concentration at the top end of the scale.
- The denominator of the equation, the AV oxygen content difference, presents a further problem, in that the mixed venous (i.e. pulmonary arterial) oxygen content has to be
measured and therefore a pulmonary artery catheter is needed to obtain the sample. Complications may arise from these catheters.

- If carefully carried out, the Fick method is accurate, but it is not practicable in routine clinical practice. Several variants of the basic method have been devised, but usually their accuracy is less good.

In clinical practice, the following adaptations of the Fick method are used for CO measurement:

(nowadays, the calculations are generally made by software built into the equipment)

1. **Dye dilution:**
   - A known amount of dye is injected into the PA & its peripherally concentration is measured by withdrawing arterial blood smoothly through a cuvette densitometer;
   - Indocyanine green is suitable due to its low toxicity and short half-life.
   - Recirculation of the dye before completion of the curve causes a second peak.
   - CO is calculated from the injected dose, the area under the curve (AUC) and its duration. (Short duration indicates high CO).
   - Arterial blood sampling can be avoided by the use of an ear-piece densitometer.

2. **Thermodilution:**
   - 5-10 ml cold saline is injected into RA through the proximal port of a PA catheter.
   - Temperature changes are measured by a distal thermistor in the PA;
   - A plot of temperature change vs time gives a similar curve to the dye curve (but without the second peak).
   - Calculation of CO is achieved using the Stewart-Hamilton equation;
   - This is the invasive method of choice when using a PA catheter.


- The thermistor probe must be matched to the CO processor, data input errors ↓ by automated computer control.
- Errors arise with tricuspid incompetence or arrhythmias so measurements should be averaged over several beats.
- Injections during the inspiratory or expiratory phases of mechanical ventilation may vary by up to 50% in calculated cardiac output.
- To minimize this error, injections are made at end-expiration.

**Limitations:**
- These methods will be less accurate if there is any intracardiac shunting.
- The size of the catheters is a limiting factor for smaller children.
- Passing a catheter into the heart can cause arrhythmias, cardiac or major vessel perforation or damage.
- The catheters can coil and become stuck.
- Inaccuracy from dye accumulation can occur with repeated measurements (dye method).
- Bleeding / thrombosis in vessel used for access.

**Variations in cardiac output curves:**
Please see referenced articles.

The above methods only provide intermittent CO measurements. In critically ill patients, continuous measurement is more useful, so a number of continuous methods (CCO) have
been devised. These are generally based on derived algorithms, but may give useful approximations, and good trends.

For example:

**Continuous thermodilution cardiac output:**
Continuous cardiac output catheters ('CCO') measure cardiac output 'continuously' by applying a randomly pulsed heating current to a filament located in the right ventricular portion of the catheter. A rapid-response thermistor analyses the resulting thermal transients with measurement of the resulting changes in blood temperature in the PA., and in conjunction with specialised hard- and soft/ware calculates the cardiac output. Measurements are updated every thirty seconds and report the average output over the preceding few minutes. Recent studies suggest that the bias and precision of CCO measurements are clinically acceptable in comparison to cardiac output determinations made using conventional thermodilution.

In children the current most used and useful method is probably the PiCCO machine.
PiCCO technology is based on a hemodynamic monitoring method, which is a combination of transpulmonary thermodilution and arterial pulse contour analysis - (Assessment of the arterial waveform and the 'area under the curve'). By “transpulmonary” is meant that the cold bolus traverses the lungs after being injected through a CVP line, and that the thermodilution curve is being measured in a systemic artery.

The method provides the following parameters:-
- **Continuous Pulse Contour Cardiac Output (PCCO)** (beat-to-beat);
- **Intrathoracic Blood Volume (ITBV),** which is a **volumetric** measure of cardiac preload;
- **Cardiac Function Index (CFI)** which reflects cardiac contractile function;
- **LV afterload** (arterial pressure and systemic vascular resistance);
- **ExtraVascular Lung Water (EVLW)** which reflects the level of pulmonary edema, if increased.
- **Stroke Volume Variation (SVV),** which is a continuous indicator of volume responsiveness (only in patients on fully controlled mechanical ventilation).

The advantage of this method is that the measurement of all these parameters, which compose a unique method for advanced hemodynamic monitoring, requires a single bolus injection of cold saline through any central venous line and a modified (thermistor-tipped) arterial catheter, eliminating the need for a Right Heart Catheter (RHC).

There remain issues of size, as the arterial catheter is generally larger than is more routinely used in small children.

**NB**

It is also possible, in older children, to insert specially designed arterial & venous lines with miniaturized arterial oxygen electrodes on their tips, giving continuous arterial oxygenation tension or mixed venous oxygen saturation SvO₂.

**References.**

- Courtman SP et al. Comparison of the bispectral index monitor with the Comfort score in assessing level of sedation in critically ill children. Int Care Med 003, 29: 2239-2246.
- Atritisch et al. Bispectral index versus COMFORT score to determine the level of sedation in paediatric intensive care unit patients: a prospective study. Critical Care 2005, 9:R9-R17