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Anaesthesia for Nurses Mini Series

Session Two: Monitoring the Anaesthetised Patient

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Anaesthesia for Nurses: Week 2

Monitoring Patients Under Anaesthesia

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Cardiovascular monitoring

Cardiovascular monitoring includes assessing indicators of adequate circulation, and adequate oxygenation. Indicators of circulation include: heart rate & rhythm, pulse rate and strength, mucous membrane colour, capillary refill time, blood pressure and central venous pressure monitoring. Indicators of oxygenation include mucous membrane colour, pulse oximetry and blood gas analysis.

Assessment of circulation

Heart rate & rhythm

The heart rate and rhythm can be counted and assessed using an oesophageal stethoscope or an electrocardiogram (ECG). Oesophageal stethoscopes may be useful as they are simple and easy to use. They allow the heart rate and rhythm to be heard from a distance when the chest is covered by a drape, they tell you the heart is beating and how fast it is beating. An irregular heart rhythm may be heard but an ECG will be required to identify the arrhythmia and some arrhythmias may not be heard on auscultation. It is important to remember that oesophageal stethoscopes and ECG's used alone provide no information on peripheral perfusion and whether cardiac output is adequate.

Bradycardia is defined as an abnormally low heart rate, <50-60 bpm in dogs and <100-120 bpm in the cat, although comparing the heart rate before and during anaesthesia ids more useful. It is quite common under anaesthesia due to the cardiovascular depressive effects of anaesthesia. Bradycardia should be treated if it is effecting the blood pressure, causing hypotension. Other causes of bradycardia include hypothermia and vagal nerve stimulation.

Tachycardia is an abnormally high heart rate. Causes include: inadequate anaestehtic depth, pain, shock, haemorrhage. Tachycardia increases myocardial oxygen demand, and can decreases cardiac output due to decreased time for the heart to fill.

Oesophageal stethoscope

- Cheap monitoring aid
- Consists of plastic tube covered at one end with a thin diaphragm
- The tube is inserted into the oesophagus until the diaphragm is level with the heart
- The other end is connected to a normal stethoscope headset
- The device detects the heart beat and **heart rate** and **respiratory rate** can be monitored

Electrocardiography (ECG)

ECGS's demonstrate the electrical activity of the heart and display the heart rate and rhythm. It is useful to detect and identify cardiac arrhythmias which are common in critically ill and trauma patients. When muscle contracts there is movement of ions in & out of the muscle cell, this generates a voltage change. The heart is no exception and the muscle is large and has a co-ordinated contraction so the voltage change is relatively large & easy to monitor, it is this electrical activity that is recorded on an ECG. If possible all trauma patients should have an ECG performed and an ECG should be performed prior to anaesthesia on any patient if the pre-operative assessment detects an unusual heart rate or rhythm, or there is pulse deficits present (Ie not a pulse for every heart beat).

It is important to remember that a normal ECG is not indicative of adequate cardiac output., therefore it should be used in conjunction with other monitoring equipment. Nurses using ECG monitoring should have an understanding of what a normal ECG trace looks like and how it is formed, and to be able to identify when an abnormal rhythm or complex is present.

The cardiac cycle

The heart is able to contract rhythmically without a constant nerve supply, although the autonomic nervous system controls the heart rate. The transmission of the electrical impulse through the heart occurs in 2 ways; Cell to cell transmission and a specialist conduction system

Cell to cell transmission

Heart muscle cells are electrically connected to their immediate neighbours
If a muscle cell depolarises (then contracts), those connected to it will follow. This creates a wave of depolarisation (then contraction) through the heart.

The specialist conduction system

This governs how the the order in which the cells depolarise and contract.
The sinoatrial node (SAN) is an area of specialist cardiac muscle in the wall of the right atrium, and it acts as the pacemaker of the heart, initiating each heartbeat. Impulses from the SAN spread across the atria. A fibrous ring found between atria & ventricles prevents the wave of depolarisation from immediately spreading to ventricles.
The atrioventricular (AV) node in the in the intraventricular septum is the only electrical connection between atria and ventricles. It is made up of slow conduction fibres crossing the insulating layer. This creates a slight pause between atrial & ventricular contractions allowing time for the ventricles to fill with blood.
Bundles of His conduct the wave of depolarisation down the septum to the apex, where Purkinje fibres conduct the impulse from the apex to the myocardium of the ventricles. This ensures the wave of depolarisation (and then contraction) travels through the ventricles from the apex upwards.

Arrhythmias

An arrhythmia is an abnormality in the electrical activity of the heart. They can effect heart rate, rhythm, force of contraction and site of origin of the heart beat. They are common in the critically ill, particularly trauma patients. Arrhythmias can be caused by: Pain, hypoxia, direct myocardial trauma (after an RTA), electrolyte imbalances, head trauma, splenic masses, GDV's, pyometras, anaesthetic drugs and hypothermia. Arrhythmias are undesirable under anaesthesia as they may reduce cardiac output by effecting how effectively the heart contracts. The abnormal electrical activity means the conduction of electrical activity does not occur in the normal way meaning the ventricles may not fill with blood properly or may not contract properly. This can result in a reduced cardiac output and reduced or absent pulse pressure. If cardiac output is significantly affected the blood pressure (and EtCO2) will fall. Ideally all trauma patients or patients with pulse deficits should have an ECG prior to anaesthesia and any arrhythmia corrected by treating the underlying cause (correcting electrolyte imbalances, administering analgesia etc) prior to anaesthesia. Patients with normal heart rhythms before induction may also develop arrhythmias under anaesthesia.

Arrhythmias under anaesthesia

Unless you are used to interpreting and monitoring ECGS start with the basics, don't dwell too much on trying to identify what the arrhythmia is. It is more important to ask yourself if the arrhythmia is causing a problem for that patient. First, don't panic, a one-off abnormal complex is unlikely to be life threatening!

Palpate the pulse to assess quality and rate, does the pulse rate match the heart rate on the ECG? Has the blood pressure or $EtCO_2$ fallen? How frequently are they arrhythmias occurring? The arrhythmia is much more serious if there are hardly any or a complete absence of normal complexes. If the blood pressure is unaffected, the pulse quality good, and there are far more normal than abnormal complexes, then it is unlikely to be serious. Always inform your vet and get them to identify the arrhythmia. Treatment of arrhythmias involves treating the underlying cause. It may be sensible to discontinue any nitrous oxide and supplement 100% oxygen if you are not already, and check the SpO_2 to rule out hypoxia as a cause. Could the animal be in pain? Or too lightly anaesthetized? Consider checking the patients electrolyte levels if possible. Consult the vet and consider administering additional analgesia. Have you given drugs which may cause arrhythmias? The answer is probably yes as most anaesthetic drugs have the potential to cause arrhythmias. The use of some anaesthetic agents such as halothane are more likely to cause arrhythmias. Halothane sensitizes the heart to circulating catecholamines. The use of drugs such as medetomidine or fentanyl which cause bradycardia may cause bradyarrhythmias such as 2^{nd} degree AV block, (this can also be seen with hypothermia). This is not normally significant, however if the heart rate is very low or AV blocks are occurring very frequently then it may be worth antagonising the medetomidine or using atropine or glycopyrrolate to increase the heart rate. Patients undergoing GDV surgery or splenectomies are prone to ventricular premature complexes (VPCs). A VPC is a complex that arises from the ventricles when they are stimulated to contract before they are meant to, due to abnormal electrical activity. Instead of following the normal conduction system from the sinoatrial node in the right atria, the electrical activity occurs prematurely in the ventricles. Whilst common if these become frequent (normally runs of 6 or more) or are affecting the blood pressure, then treatment with lidocaine should begin. If left untreated they may significantly effect cardiac output and can lead to ventricular tachycardia which is life threatening. Be prepared. Discuss with your vet prior to anaesthesia if possible, what to do and how much lidocaine to administer if this occurs, have some lidocaine to hand and have the dose already calculated.

Peripehral pulse rate and rhythm

Peripheral pulses should be palpated rather than a central pulse such as the femoral because peripheral pulses are more sensitive to changes in intravascular volume. The pulse strength can be compared to pre-anaesthesia. The pulse pressure felt when a pulse is palpated is the difference between systolic and diastolic blood pressure. A strong peripheral pulse indicates good peripheral pulse quality. A reduced/poor pulse quality indicates peripheral vasoconstriction due to: ‘shock’, haemorrhage, hypothermia, or fear. The pulse rate should be checked to ensure it matches the heart rate to check for pulse deficits seen with arrhythmias.

Mucous membrane (mm) colour & capillary refill time (CRT)

Examining the mm colour gives a crude assessment of both tissue perfusion and oxygenation (see later). It is normally assessed by observing the gingiva, however in pigmented animals the vulva/prepuce or conjunctiva can be examined. Pale mm indicate vasoconstriction due to shock, anaemia or haemorrhage, however it should be used in conjunction with assessment of other parameters as other factors such as body temperature and fear may confuse assessment.

Capillary refill time

Capillary refill time is the rate of return to colour of oral mucous membranes after application of gentle digital pressure. It gives an indication of peripheral perfusion. A prolonged CRT (> 2 seconds) indicates that perfusion to that area is reduced. This could be due to vasoconstriction, hypovolaemia, haemorrhage or too deep anaesthesia.

Arterial blood pressure

Since cardiac output cannot easily be measured clinically, other methods of estimating blood flow to the tissues must be used. Blood pressure provides the driving force for tissue perfusion.
Blood pressure = cardiac output X systemic vascular resistance
Cardiac output = Heart rate X Stroke volume
What these mean is that blood pressure is dependent on cardiac output and systemic vascular resistance (Vasodilation/vasoconstriction). Cardiac output is dependent on heart rate and stroke volume (the volume of blood pumped out in one contraction). If either one of these decreases then the cardiac output may decrease. In healthy patients there are compensatory mechanisms that prevent a fall in blood pressure, so for example if the cardiac output falls then systemic vascular resistance will increase (vasoconstriction) to compensate and maintain blood pressure. However in sick patients these compensatory mechanisms may not be able to function normally. When the mean arterial blood pressure (MABP) is maintained in the range of about 60 to 120 mmHg, blood flow to the brain and kidney is autoregulated via local mechanisms that maintain adequate perfusion. When MABP falls below about 60 mmHg, tissue perfusion is reduced. Hypotension can result in lactic acidosis and hypoxia due to inadequate tissue perfusion. The kidneys and brain are especially susceptible to this ischaemic damage. Blood pressure can also be used as a guide to depth of anaesthesia with increasing blood pressure potentially indicating lightening anaesthetic depth and decreasing blood pressure deepening anaesthesia. If hypotension occurs, depth of anaesthesia should be decreased and IV fluids administered to effect. The use of drugs which cause vasoconstriction such as medetomidine will give a high blood pressure reading although tissue perfusion may not be adequate. There are two ways to monitor blood pressure; Direct (invasive) and indirect (non-invasive)

Invasive (Direct) blood pressure

Is the most accurate way of monitoring blood pressure but due to the cost, technical difficulty and risks, it is rarely performed in general practice. If the skill and equipment exist in your practice it may be useful for critically ill patients. A catheter must be placed directly into the artery (normally the dorsal metatarsal) which is then connected to a calibrated electrical transducer via saline filled non compressible tubing. This allows the arterial pressure waveform to be displayed on the screen. It provides continuous, real time measurements of the systolic, diastolic and mean blood pressure and displays the waveform which can provide useful information about the cardiovascular system. The catheter must be placed aseptically, flushed frequently to prevent thrombus formation, and there is a risk of infection and haemorrhage/haematoma formation.

Non-invasive blood pressure monitoring

Oscillometric technique
This is an automated technique found in multiparameter monitoring machines, where a cuff is placed over an artery. The cuff is inflated to a pressure at which arterial blood flow is occluded, and then gradually deflated. Pressure within the cuff oscillates when the blood flow resumes and this is detected by the machine. Systolic, mean and diastolic pressures are measured. The machine can be programmed to cycle at regular intervals. The oscillometric technique may be inaccurate on smaller patients, in the presence of vasoconstriction, tachycardia, bradycardia, hypotension, hypertension and arrhythmias. In these cases the use of a Doppler probe may be more useful.

Doppler technique (Doppler ultrasonic flow detection)

The Doppler technique measures systolic blood pressure and involves the use of piezoelectric crystals which transmit and receive sound. The technique relies on the fact that the frequency of sound reflected from moving tissues (arterial blood) differs from that transmitted from the crystal. Many people find it useful as it provides an audible pulse and will still give a systolic blood pressure in cases where the oscillometric technique normally won't (bradycardia, arrhythmias and hypo/hypertension). It is certainly recommend to place a doppler probe and cuff when anaesthetising critically ill patients if you have one in your practice.

Whichever type of non-invasive blood pressure technique you use, the selection of a correct size cuff and correct cuff placement is important to ensure accurate readings. The width of the cuff should be 40% of the circumference of the limb and the bladder of the cuff should be placed over the artery. The cuff should be wrapped around the limb firmly, neither too tight nor too loose, and tape should not be wrapped all the way round the cuff as this will prevent the cuff from inflating properly. The cuff should ideally be placed at heart level. If the cuff is too loose, lower than heart level or too small, the reading will overestimate the true blood pressure. If the cuff is too tight, too big or higher than the heart, then the blood pressure machine will underestimate the real blood pressure.

Hypotension

Hypotension is usually defined as a mean arterial pressure of less than 60 mmHg. Hypotension can be due to decreased cardiac output, decreased systemic vascular resistance (vasodilation) or a combination of the two. Hypotension under anaestehsia can occur for many reasons which lead to one of the above causes.

Deep anaesthesia

Inhalational agents cause profound cardiovascular depression. Isoflurane and sevoflurane cause marked vasodilation which reduces systemic vascular resistance and can cause hypotension. They also cause a drop in cardiac output although this effect is not as pronounced as with halothane.

Haemorrhage

Intraoperative haemorrhage causes a reduced cardiac output due to a reduced preload (the amount of blood returning to the heart) and should be addressed with fluid therapy. If using crystalloids approximately 4 times the volume of blood loss will be required as the fluid is rapidly redistributed to the extravascular fluid compartments. Communication with the surgeon is vital to spot blood loss before it becomes catastrophic. Working out the patients circulating blood volume prior to surgery will be useful so you can put the amount of blood loss into perspective in relation to the patients size. Use approximately 88mL/kg for dogs and 66mL/kg for cats. This enables you to calculate the blood loss as a percentage of the body weight. It has been suggested that blood loss of < 15% can be replaced with crystalloid, blood loss of 15-20% with a colloid and > 20% with whole blood. Animals are able to compensate for a degree of blood loss. Compensatory mechanisms occur to counteract the reduction in circulating volume, the heart rate will increase to increase cardiac output and vasoconstriction may occur to maintain blood pressure. A drop in blood pressure is a late change with haemorrhage due to these compensatory mechanisms. The speed of blood loss and the health of the patient influences how well a patient a patient is able to 'cope' with blood loss, so the patients clinical signs and vital parameters should be used to guide the fluid replacement.

Decreased cardiac output

Cardiac output is dependent on stroke volume and heart rate, therefore a reduction in either can decrease cardiac output. Stroke volume can be reduced by a reduced cardiac contractility or decreased stroke volume. Reduced contractility can occur due to heart disease, drugs, such as halothane, or severe hypothermia. Stroke volume can be reduced by a decreased preload due to reduced venous return (the blood returning to the heart). Factors reducing venous return include; hypovolaemia , haemorrhage, IPPV, dorsal recumbency in pregnant females, large abdominal masses or obese patients.

Bradycardia, severe cardiac arrhythmias

Bradycardia can also reduce cardiac output and therefore blood pressure. Bradycardia can be hard to define as it depends on the species, breed and fitness of the patient, but generally is defined as a heart rate of < 50-60 bpm in dogs and < 100 bpm in cats. Rather than dwelling on the values to define bradycardia, a more useful way of looking at it is whether the bradycardia is affecting the blood pressure, if it is, then it needs treating. Drugs such as alpha 2 agonists like medetomidine can cause bradycardia and antagonizing them could be considered. Hypothermia causes bradycardia and reduced cardiac contractility so body temperature should be monitored and steps taken to minimize hypothermia. Bradycardia can be treated with an anticholinergic (unless the cause is hypothermia) such as atropine or glycopyyrrolate, although they can cause tachycardia.

Sepsis

Sepsis can occur due to many diseases including; GDV, perforated intestines due to foreign bodies or pancreatitis. Sepsis causes myocardial depression, reduced cardiac contractility and vasodilation due to inflammatory mediators which are released. These patients often are also hypovolaemic due to third space fluid loss into the abdominal cavity. Septic patients require complex management. Ideally replacement of lost fluid with aggressive fluid therapy will occur prior to anaesthesia. Minimising anaesthetic time is important to minimize organ compromise and using a balanced anaesthetic protocol to reduce isoflurane requirements is crucial.

Cardiac disease

Heart disease can reduce cardiac contractility which may reduce cardiac output. If cardiac disease is known then caution should be taken with treating with fluid therapy.

Central venous pressure (CVP)

Central venous pressure is the pressure with the cranial vena cava (the intrathoracic portion of the vena cava). CVP reflects the volume of blood returning to the heart and provides information about intravascular blood volume and cardiac function. It can help guide adequacy and quantity of fluid therapy so may be useful to monitor in critically ill patients. The normal value in anaesthetised patients is 2-7 cm H_2O, and serial measurements should be taken as a single measurement gives little information. Measuring CVP involves the aseptic placement of a Jugular catheter with the tip sitting in the cranial vena cava. Most multiparameter monitors have electrical trandsducers suitable for monitoring CVP and arterial blood pressure and the transducer should be placed at heart level and connected to the catheter via saline filled non-compliant tubing.

Indicators of oxygenation

Assessment of mm colour gives a crude estimate of oxygenation as cyanotic (blue/purple) mm colour indicates a very low blood oxygen concentration (hypoxaemia). Cyanosis may be seen in respiratory arrest, inadequate oxygen supply or severe pulmonary disease.
In order to understand parameters used to assess oxygenation an understanding of the basic physiology of oxygen transport is useful. Oxygen is carried in the blood in two forms: free, dissolved in the plasma (PaO2) (around 1-2%) and bound to the haemoglobin (Hb) in the red blood cells. Therefore oxygen content in the blood is dependent on the haemoglobin (Hb) concentration, the percentage of Hb saturated with oxygen (SpO2) and the amount of oxygen dissolved in the plasma (PaO2). Oxygen delivery to tissues is dependent on the cardiac output and the oxygen content in the blood.

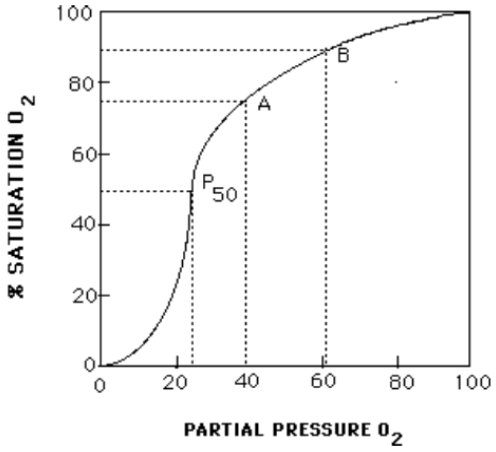
Pulse Oximetry

A pulse oximeter tells you the percentage of haemoglobin saturated with oxygen and gives you an indication of the adequacy of oxygen transport. Oxygen delivery to tissues is also dependent on haemoglobin concentration and cardiac output, so it is important to remember that anaemic patients may have a reduced oxygen carrying capacity due to a low haemoglobin but the SpO2 will be normal as long as the lungs are working.

Pulse oximeters are widely used in practice as their price has fallen. They are non-invasive, easy to use and provide continuous immediate monitoring of pulse rate and arterial haemoglobin saturation. However, it is important to understand what it they demonstrate and their limitations in order to utilise it to its full potential.

Patients with normal lungs, receiving 100% oxygen at adequate flow rates are extremely unlikely to desaturate, however pulse oximeters are particularly useful for patients with lung disease, those undergoing a thoracotomy, in the recovery period when the patient is breathing room air and to aid detection of equipment problems such as inadequate oxygen supply. It is important to remember that the pulse oximeter is a late indicator of hypoxaemia, especially if the patient is breathing 100% oxygen. Serial blood gas analysis on critical patients undergoing a thoracotomy where possible should be performed. Pulse oximetry is also useful to measure the pulse rate and to detect equipment problems such as inadequate provision of oxygen. In most patients pulse oximeters will detect desaturation before cyanosis is detectable. SpO_2 is related to arterial partial pressure of oxygen (PaO_2) by a sigmoid relationship (the oxyhaemoglobin dissociation curve, see below). This means that the SpO_2 will remain high as the PaO_2 falls and the SpO_2 will only drop when the PaO_2 has already reached very low levels. If the SpO_2 falls to 90%, the patient is borderline hypoxaemic.

The oxygen dissociation curve



The visible pulse waveform (plethysmograph trace) is also useful in the presence of arrhythmias to detect pulse deficits.

Limitations of pulse oximetry

There are several situations where pulse oximeters may fail to give a reading, or may give inaccurate ones:

1. Vibration / patient movement

It is much more difficult (though still possible) to obtain accurate pulse oximeter readings on conscious patients than those that are unconscious, simply due to movement artefact, as this interferes with the machine's ability to detect pulsatile blood flow in the tissues.

2. Vasoconstriction

Marked peripheral vasoconstriction - as may be seen following administration of alpha-2 agonists, such as medetomidine - may prevent some pulse oximeters from obtaining readings. Better quality devices can generally still obtain a signal, although this cannot be guaranteed.

3. Visible light

Light sources - particularly if pulsatile (e.g. fluorescent strip lights) - can interfere with the ability of the pulse oximeter to get a signal. This is less common with newer machines, as shielding of the probe has improved over the years, but may still occur if the probe is partially hanging off the tissue.

4. Calibration

Calibration of pulse oximeters is achieved by using human volunteers and progressively desaturating them by making them breathe increasingly hypoxic gas mixtures. Because it is considered unethical to desaturate these volunteers to a SpO2 of <80%, readings below this value on the pulse oximeter are extrapolated, i.e. accuracy may be imprecise. However, potentially it doesn't matter too much if the patient's SpO2 is 49% or 67%, because both values imply a serious problem.

5. Pigmentation
It may be difficult to obtain a good signal if the pulse oximeter probe is placed on a heavily pigmented area. This can be problematic in breeds such as Shar Peis and Chows, where the tongue and oral mucosa are often black. A signal can sometimes be obtained in these animals by attaching the probe to the vulva or prepuce, as this is often less heavily pigmented.
6. Pulsatile veins
Although not a common problem, the pulse oximeter can be confused if there is pulsatile flow within veins, and may give falsely low saturation readings. This can sometimes occur with severe tricuspid regurgitation or 3 (complete) atrio-ventricular blockade.
7. Dyshaemoglobinaemias
There are 2 main abnormal forms of Hb that can lead to inaccurate pulse oximeter readings. Methaemoglobinaemia may occur following local anaesthetic toxicity (particularly prilocaine), but is also seen in cats with paracetamol intoxication. This form of dyshaemoglobin tends to make the pulse oximeter read towards 85%, and no conclusions regarding oxygenation can be made using pulse oximetry in this setting. The second significant dyshaemoglobin is carboxyhaemoglobin , which may be seen in patients following carbon monoxide intoxication (e.g. animals trapped in house fires). Carboxyhaemoglobin tends to push pulse oximeter values towards 100%, as it has a similar light absorption spectrum to oxyhaemoglobin. Again, pulse oximetry is an unsuitable means of assessing patient oxygenation if carboxyhaemoglobin is
present.

Pulse oximeters are most useful in recovery when the patient is breathing room air not 100% oxygen, particularly brachycephalic patients or others with URT disease- laryngeal paralysis, myasthenia gravis etc and patients recovery from thoracotomy or with lung disease. It is important to remember that the SpO_2 reading is only reliable when the pulse oximeter is displaying a heart rate or plethysmograph trace that corresponds to the pulse rate (check by palpation).

Conclusion
Pulse oximetry is a useful monitoring tool in both awake and anaesthetised patients. However, it does have limitations, and these must be understood to appreciate the value of the information it is providing.

Respiratory Monitoring

Respiratory monitoring involves assessing the adequacy of ventilation and gaseous exchange. It is important that the respiratory system is monitored closely under anaesthesia as anaesthetic agents cause dose dependent respiratory depression. Close monitoring also detects problems with the ETT such as occlusion or kinking as well as equipment malfunction. The reason for anaesthesia and the patients' disease processess may also impact the respiratory system under anaesthesia.

Ventilation can be assessed by observing the patients respiratory rate, depth and character. The rate can be counted by observing the chest movement or the rebreathing bag. The respiratory character should be assessed by observing the chest throughout anaesthesia. There should be good chest wall and diaphragmatic movement with minimal abdominal effort. Increased respiratory effort may indicate ETT occlusion. A normal inspiration lasts about 1 second with expiration lasting about 2-3 seconds, creating a normal inspiratory to expiratory ratio of 1:2 or 1:3. A Wrights respirometer can be used to accurately measure tidal volume and may be useful in patients with reduced lung volumes prior to setting ventilator settings.

Tidal volume is the amount of air breathed out in a normal breath and is about 10-15 mL/kg. Tidal volume will decrease under anaesthesia and atelectasis occurs.

Apnoea monitors are relatively common in practice and consist of a sensor placed between the ETT and the breathing system. They detect the movement of warm gases through the breathing system and provide an audible beep with each breath. They provide no information about tidal volume or adequacy of ventilation and can be affected by cardiogenic oscillations that causes gas movement in the trachea. They simply tell you if the patient is breathing or not.

Capnography

Capnography provides a non-invasive method that permits the assessment of the adequacy of patient ventilation, systemic metabolism, cardiac output and pulmonary perfusion in a variety of clinical situations, such as during anaesthesia, when effects of drugs and inhalants are likely to cause respiratory depression, or during long-term ventilatory assistance as with the use of a mechanical ventilator. Normal $ETCO_2$ values, in non-anaesthetised patients, are 35- to 45 mm Hg. End-tidal CO_2 values above 50 mm Hg indicate inadequate ventilation, and ventilatory assistance via manual or mechanical means by be required. The highest $ETCO_2$ permissible should be 60 mm Hg.

Capnography is also superior over pulse oximetry for the prompt identification of apnoea and airway issues, since changes in the percentage of haemoglobin saturated with oxygen (SpO_2) will be delayed as compared to the instantaneous changes that occur with $ETCO_2$ when the next breath fails detection. When alveoli are not perfused, carbon dioxide is unable to diffuse out of the bloodstream. But as blood flow improves and alveoli are perfused, carbon dioxide can then be excreted. Therefore, an abrupt decrease in $ETCO_2$ can be an early and reliable indication of an impending cardiovascular collapse or cardiac arrest. Since delivery of carbon dioxide from the lungs requires blood flow, cellular metabolism, and alveolar ventilation, the presence of $ETCO_2$ can also be used to assess the effectiveness of cardio-pulmonary cerebral resuscitation (CPCR) efforts (Jandrey, 2006).

Carbon Dioxide Physiology

Carbon dioxide is transported in the body in 3 forms: after conversion in the red blood cells, 60- to 70% is transported as bicarbonate ion, another 20- to 30% is transported while bound to proteins, and the remaining 5- to 10% is dissolved in plasma. The latter is what is actually measured during blood gas analysis and is known as the arterial partial pressure of carbon dioxide ($PaCO_2$). End-tidal carbon dioxide ($ETCO_2$) is the result of expired gases from the alveoli. The site of gas exchange occurs in the alveolar capillary beds lying between the blood and air within the lungs. Under normal circumstances, $ETCO_2$ typically underestimates the $PaCO_2$ by a clinically insignificant 2- to 5 mm Hg. Therefore, because of the extremely close proximity of gas exchange between the alveoli and pulmonary capillaries, $ETCO_2 \approx PaCO_2 \approx$ alveolar CO_2 ($PACO_2$) (Reuss Lamsky, 2010).

Carbon Dioxide Monitoring

End-tidal CO_2 is the partial pressure of carbon dioxide in the expired air obtained at the end of expiration. This value approximates that of alveolar air, assuming that:

1. Capillary blood and alveolar gas CO_2 are in equilibrium
2. End-tidal CO_2 approximates the time weighted average of the ventilation weighted $PaCO_2$

3. Ventilation/Perfusion (V/Q) mismatch does not exist
4. Tidal volumes are large enough to displace dead space
5. Fresh gas flow is low enough to prevent dilution, and sample aspiration is low enough as to not entrain air or interfere with patient ventilation

End-tidal CO_2 is a product of three major determinants:

1. The rate of CO_2 production by the tissues
2. The rate of exchange of CO_2 from the blood to the alveoli
3. The rate of CO_2 removal by alveolar ventilation.

End tidal CO_2 can provide therefore provide information regarding (1) metabolism, (2) circulation, and (3) ventilation. Most capnographs used in clinical monitoring use infrared absorption spectroscopy to determine the concentration of carbon dioxide in the expired air. Infrared light, at wavelengths absorbed by carbon dioxide, is passed through the expired gas sample and the concentration is determined according to the Lamber-Beer law. Capnography is a much more straightforward technology than pulse oximetry in that the sample is removed from the patient and is not subject to corrections for patient's background interference (Jandrey, 2006).

Technical Considerations

Capnometry is the measurement of CO_2 at the airway opening during the ventilatory cycle ($PetCO_2$). The capnometer displays the numerical value for PCO_2. Capnography is the waveform display of CO_2 as a function of time or volume. A device that measures CO_2 and displays a waveform is a capnograph. The waveform displayed by a capnograph is called a capnogram. Capnometers may use infrared (most common), Raman scattering, mass and colorimetric spectroscopy for measurement of CO_2.

Mainstream or sidestream capnometry describes the location of the measurement chamber or airway sampling site.

Mainstream capnometers place the measurement chamber within the airways. This allows for an almost instantaneous measurement of CO_2. Some drawbacks of mainstream capnometry include: 1) they are easily damaged, 2) their presence increases dead space, 3) they are difficult to use in spontaneously breathing patients, and 4) water condensation often occurs on the sensor (Figure 1).

Figure 1: Mainstream capnometer chamber

Sidestream capnometers sample air aspirated out of the airway through fine bore tubing to a measurement chamber outside the device (Figure 2). An advantage of sidestream analysis is that the units often measure other gases (i.e., O_2, anaesthetics). Slight delays in measurement may occur due to movement of the sample through the tubing. Secretions from the airway may easily obstruct the tubing. Neither is clearly superior, and the choice between them is most often a personal preference.

Figure 2: Sidestream capnometer port.

Interpretation of the Capnogram

The normal capnogram (see Figure 3) can be divided into four main phases:

Phase I - this is normally the flat baseline segment of the capnogram. The first part of this phase is when inspiration occurs. At the end of this phase, the direction of gas flow reverses as expiration occurs. During early expiration, the exhaled gas comes from anatomic dead space, this gas has not undergone any gas exchange, and as a result the gas from this area is identical to inspired gas (i.e. contains no CO_2).

Phase II – this is the upstroke of the waveform. This corresponds to the part of exhalation where CO2 containing alveolar gas starts to be exhaled in a mixture with gas from the anatomical dead space. As expiration continues, the expired gas is composed of rapidly increasing proportions of alveolar gas, and the CO2 levels quickly begin to rise.

Phase III – This phase is the plateau of the capnogram. At this point PCO2 is normally almost constant as alveolar gas is exhaled. Expiration actually ends partway through this phase and is usually followed by a pause. During this pause, PCO2 normally remains constant on the capnogram, despite the fact there is no gas flowing in or out of the patient. This is because there is expired alveolar gas remaining stationary within the region of the breathing circuit from which the gas is being sampled. This part of the plateau may be cut short by small tidal volumes, high fresh gas flow rates, and/or high gas sampling rates. The angle between phases II and III of the capnogram, is known as the alpha angle and is normally around 100-110°.

Phase IV – this is the rapid downstroke on the capnogram which corresponds with inspiration. During this phase, fresh inspired gas (which should be CO2 free) passes the sampling port as it is inspired into the lungs. The angle between phases III and IV is know as the beta angle and is normally around 90°.

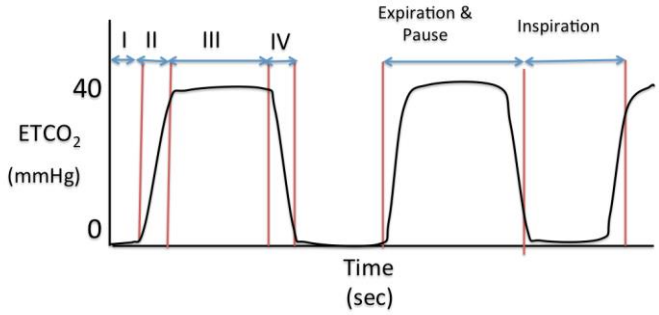


Figure 3: Normal capnogram

Aberrations in the capnographic waveform may occur. Therefore it is important to read the $PetCO_2$ and also assess the capnogram.

Abnormal phase I – If the capnogram does not return to baseline during inspiration, and this is not thought to be due to a slow response time of the analyser (i.e. the waveform is a normal shape) then the patient must be inhaling CO2. Common reasons for this abnormality include in adequate fresh gas flow rates when using non-rebreathing systems, e.g. Bain system, exhausted CO2 absorbed within rebreathing system, e.g. Circle system, or a malfunctioning inspiratory valve in a circle system (Figure 4).

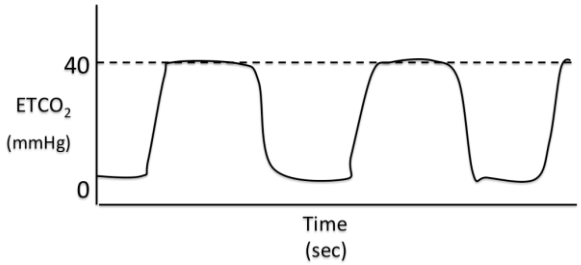


Figure 4: Rebreathing on a capnogram

Abnormal phase II – When using sidestream capnographs, gas sampling rate can affect the rate of the capnogram. Slow sampling rates will decrease the slope of phase II, shorten the alveolar plateau, and decrease the slope of phase IV. This delay in response time will commonly cause an increase in the alpha and beta angle of the capnogram. If the slope of phase II is decreased in the absence of delayed equipment response time, it suggests a slow expiration time. Causes of slow expiration include partially obstructed (e.g. mucus plug) or kinked endotracheal tubes, physical conditions which cause a narrowing of the airway (Figure 5).

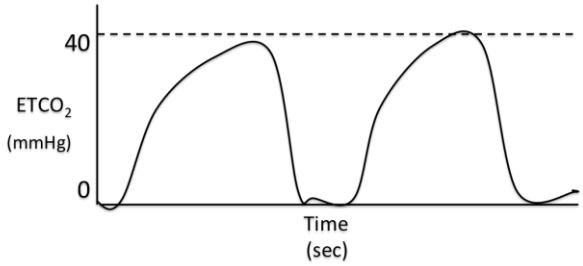


Figure 5: Slow expiration time on capnogram

Abnormal phase III – in healthy, conscious patients, peak expiratory ETCO2 values are only a few mmHg lower than PaCO2. A normally shaped capnograph with an elevated alveolar plateau indicates hypoventilation (Figure 6). In anaesthestised, or sedated, patients this is not an uncommon finding and it can be common cause of hypoxaemia.

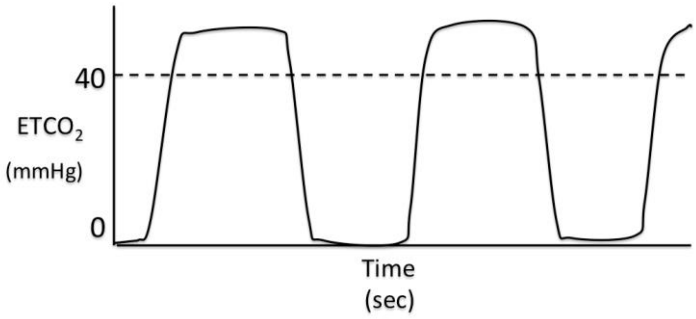


Figure 6 Hypoventilation

A normally shaped capnogram with a lower than normal plateau, may be due to hyperventilation (Figure 7). If the patient is being manually or mechanically ventilated then the rate of ventilation/ventilator settings should be evaluated. Other causes for a lower than normal alveolar plateau include decerased CO2 production (hypothermia) or decreased delivery of CO2 to the lungs (low cardiac output). Ideally blood-gas analysis should be performed when faced with persistent low ETCO2 levels with no obvious cause. The existence of alveolar dead space (ventilated but underperfused areas of the lung) which may occur secondary to pulmonary thromboembolism, creates a situation in which peak expired CO2 levels are substantially lower than PaCO2 measurements. Underperfused alveoli will not have participated in gas exchange and so contain gas identical in composition to inspired gas, which is normally CO2 free. During expiration, this gas mixes with the gas from perfused alveoli, thereby diluting the ETCO2 in the expired gas.

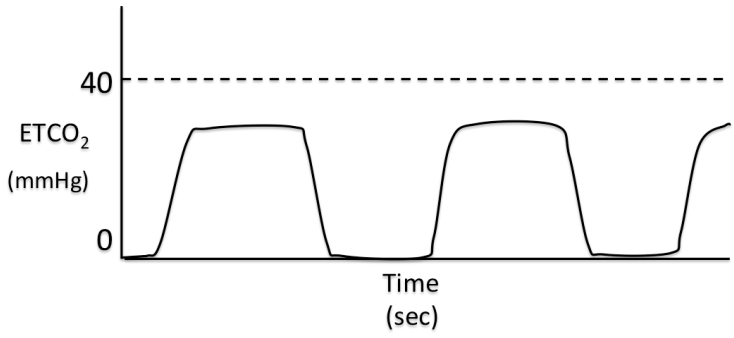


Figure 7 Hyperventilation

Equipment failure can also result in abnormally low alveolar plateaus. For example leaks in the gas sampling line, so therefore constant ‘sampling’ of room air, resulting in dilution of the exhaled gas and a falsely low ETCO2 measurement.

The normal alveolar plateau is roughly horizontal (see figure 3). Artifactual dips and bumps in the plateau may result from pressure on the thorax of an anaesthetised patient, resulting in gas moving in and out of the lungs. If an patient is being mechanically ventilated, spontaneous ventilator efforts may be interspersed amongst mechanical breaths, resulting in dips or clefts in the alveolar plateau.

Causes of these respiratory efforts should be investigated and may include: insufficient anaesthetic depth, inadequate mechanical ventilation, hypoxaemia, inadequate analgesia and hyperthermia (Figure 8).

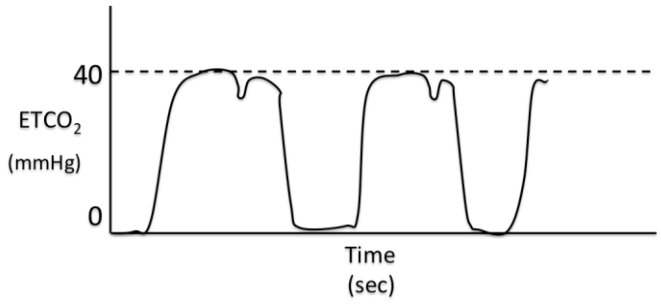


Figure 8 Spontanous respiratory efforts during mechanical ventilation

Cardiogenic or cardiac oscillations are undulations in the capnogram that are synchronous with cardiac contractions. Contraction of the right ventricle and filling of the pulmonary vasculature, results in the expulsion of a small volume of air from the lungs with each beat. This is a common and inconsequential finding in anaesthetised patients (Figure 9).

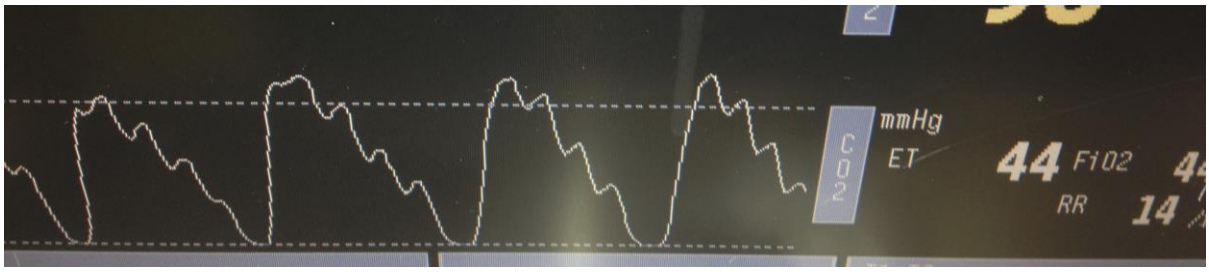


Figure 9 Cardiogenic oscillations.

Abnormal phase IV – Normally the capnogram should return to baseline from the alveolar plateau, thereby creating a beta angle of around 90°, this occurs as fresh gas is inspired thereby replacing CO_2 containing gas at the sampling site. If the slope of this phase is reduced (i.e. increased beta angle) then either inspiration is not occurring normally or there is CO2 in the inspired gas. This could occur with inadequate fresh gas flow rates on non-rebreathing systems, or a malfunctioning inspiratory valve on a rebreathing system, e.g. Circle (figure 10).

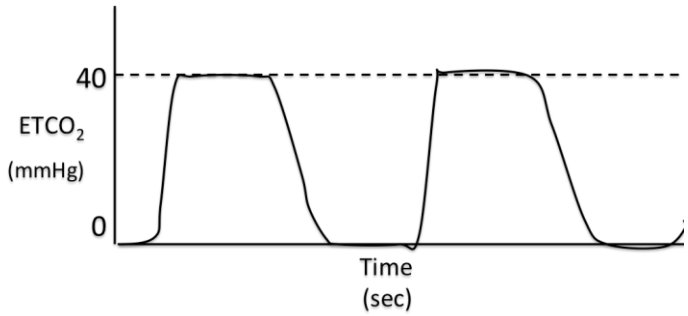


Figure 10 – Increased beta angle, indicating slow inspiration, CO2 in inspired gas or malfunctioning inspiratory valve on rebreathing system.

Step by Step Guide to Capnogram Interpretation

1. Is there a regular waveform present which indicates ventilation?
2. Does the waveform return to the baseline (zero) or is there evidence of rebreathing of CO2 (elevated baseline)?
3. Is the angle of the upstroke normal or is there evidence of slow expiration (slanted upstroke) – see figure 5.
4. Is the alveolar plateau normal or is there evidence of uneven emptying (slanted plateau) or interruption of the expiratory period by inspiratory efforts (clefts in plateau) see figure 10?
5. Are the ETCO2 values within an acceptable range, and are they consistent with the patients respiratory parameters?
6. Is the angle of the downslope normal, or is there evidence of slow inspiration or rebreathing (slanted downstroke)?

   (Barter, 2012)

Conclusion

Capnography is a useful, non-invasive technique for the continuous assessment of ventilation and perfusion in patients. Capnography is most commonly used in anaesthetised patients but can also be a useful modality in conscious patients for non-invasive PCO_2 monitoring. It is not just the ETCO2 values which provide us with information but also the waveforms which can give us a huge amount of information about a patient's condition, and provide an early warning sign to serious airway and perfusion issues.

Blood gas analysis

Is a definitive guide to pulmonary function. It provides information on the carbon dioxide and oxygen parital pressures in arterial (or venous) blood, and provides information on the acid-base status of the patient. It is not widely used, or necessary in general practice, however may be useful in certain patients if available. Arterial samples are normally taken from a peripheral artery such as the dorsal metatarsal artery and must be taken anaerobically into a heparinised syringe and then pressure applied to the site.

Temperature

Core temperature will drop during anaesthesia due to impairment of thermoregulation, exposed viscera, inhibition of shivering, ceased skeletal muscular activity and the dilation of blood vessels. Animals most at risk are those with high ratios of surface area to volume i.e. neonates, small furries and those with already impaired thermoregulatory centres i.e. very young, or geriatric patients. It is therefore, advisable to try to maintain normal body temperature and to achieve this body temperature needs to be monitored throughout anaesthesia.

A recent large scale epideimiological study demonstrated that only 11% of patients had their body temperature measured during the recovery period. This is pretty shocking as everyone has access to a thermometer and it only takes a few seconds. Simple, easy measures such as using bedding, bubble wrap, and 'hot hands' can be used to prevent hypothermia. Hypothermia under anaesthesia and in the recovery period is significant because it has a central depressant effect and reduces the MAC (therefore if left unnoticed on the same vaporizer setting the patient would get progressively more deeply anaesthetised), it prolongs drug metabolism and recovery from anaesthesia, causes bradycardia and arrhythmias,

effects cardiac contractility. it can interfere with normal haemostasis, reduce ventilation and it leads to shivering in the recovery period. Ideally core temperature should be monitored by the use of a thermistor (temperature probe) placed into the oesophagus or nasopharnyx. Most multiparameter monitors have a thermistor with them to allow continual core temperature monitoring, however if these are not available a simple mercury or digital rectal thermometer will be useful, even though it is not measuring core temperature. Reduced body temperature causes delayed/prolonged recovery from anaesthesia, a fall in cardiac output, decreased ventilation and an increase in blood viscosity; in severe cases it may lead to hypothermia and even death from cardiac arrest! Rectal mercury thermometers can be used however, flexible thermistor probes are preferable. They can be inserted into the rectum or down the oesophagus to the base of the heart and they produce a read out of core body temperature.

Cleaning & maintenance

The monitoring equipment, ECG cables, pulse oximeter probe, blood pressure cuffs etc should be cleaned between patients. Alcohol wipes are useful for this as the alcohol will evaporate. Care should be taken when using other disinfectants such as Trigene that anything such as pulse oximeter probes coming into contact with the patients skin or mucous membranes are wiped or rinsed with water before use otherwise irritation or even chemical burns may occur. Ideally, at the end of each day the equipment should be washed in a bucket of disinfectant such as Trigene suitably diluted. Most parts of the probes and cables can be immersed as long as electrical connections are not immersed. For sidestream capnographs it is important that the ends of capnograph sampling lines are not immersed and that the machine is not left on while washing, as the capnograph pump will draw water up into the sampling line and into the machine, damaging it. Ensure you know which parts of the equipment can be immersed in water to clean and which parts cannot. Again check with the supplier if in doubt. Once cleaned keep all cables neatly coiled and tidy.
It is important that if you are expected to use the equipment to monitor animals under anaesthesia that you know how the equipment works. If necessary ask for a demonstration from the supplier, read the instruction manual, and keep the phone number of the supplier somewhere to hand if there are any problems. If you are faced with an equipment problem, do not panic, work through these discussed steps logically and remember you can still adequately monitor patients under anaesthesia using your eyes, ears and brain!