**ARTERIAL BLOOD GAS COMPETENCY PACK**

This pack contains:

* Purpose and objectives
* A work book
* A competency assessment
* An action plan
* Theoretical knowledge should be gained using the ABG educational package, and should be tested using the competency sheet provided
* The theoretical tester should be an anaesthetic registrar or above
* The practical tester should be a member of medical or nursing staff who is already proficient in this practice.
* The practical element will be assessed using the competency document provided
* Further supervised practical assessments should continue to be demonstrated by the learner.
* A minimum of 6 supervised practices is suggested prior to undertaking this skill unsupervised.

The purpose of this competency package is to educate the patient care providers on:

* The basic principles of acid-base balance
* A systematic approach to the interpretation of arterial blood gases
* To perform and be aware of dangers and complications of this invasive technique
* To safely work towards competence

On completion of this competency the learner should be skilled and knowledgeable in:

* The physiology involved in the acid/base balance of the body.
* The roles of PaO2, pH, PaCO2 and bicarbonate in maintaining acid/base balance.
* The causes and treatments of Respiratory Acidosis, Respiratory Alkalosis, Metabolic Acidosis and Metabolic Alkalosis.
* Identifying normal arterial blood gas values and interprets the meaning of abnormal values.
* Interpreting the results of various arterial blood gas samples and suggestive how treatment may be changed to improve abnormal results
* Identify the relationship between oxygen saturation and PaO2 as it relates to the Oxyhaemoglobin dissociation curve.
* Interpret the oxygenation state of a patient using the reported arterial blood gas PaO2 value.





**INTERPRETATION OF THE ARTERIAL BLOOD GAS**

**SELF-LEARNING PACKAGE AND REFERENCE GUIDE.**

**PURPOSE**

The purpose of this self-learning package is to educate patient care providers on the basic principles of acid-base balance, as well as to provide a systematic approach to the interpretation of arterial blood gases.

**OBJECTIVES**

After completing this package, the learner should be able to:

* Describe the physiology involved in the acid/base balance of the body.
* Compare the roles of PaO2, pH, PaCO2 and Bicarbonate in maintaining acid/base balance.
* Discuss causes and treatments of Respiratory Acidosis, Respiratory Alkalosis, Metabolic Acidosis and Metabolic Alkalosis.
* Identify normal arterial blood gas values and interpret the meaning of abnormal values.
* Interpret the results of various arterial blood gas samples.
* Identify the relationship between oxygen saturation and PaO2 as it relates to the oxyhemoglobin dissociation curve.
* Interpret the oxygenation state of a patient using the reported arterial blood gas PaO2 value.

**INTRODUCTION**

Arterial blood gas analysis is an essential part of diagnosing and managing a patient’s oxygenation status and acid-base balance. The usefulness of this diagnostic tool is dependent on being able to correctly interpret the results. This self-learning package will examine the components of an arterial blood gas, what each component represents and the interpretation of these values to determine the patient’s condition and treatment.

**ACID-BASE BALANCE**

**OVERVIEW**

The pH is a measurement of the acidity or alkalinity of the blood. It is inversely proportional to the number of hydrogen

Ions (H+) in the blood. The more H+ present, the lower the pH will be.

Likewise, the fewer H+ present, the higher the pH will be. The pH of a solution is measured on a scale from 1 (very

acidic) to 14 (very alkalotic). A liquid with a pH of 7, such as water, is neutral (neither acidic nor alkalotic).

ACIDIC =1

NEUTRAL= 7

ALKALOTIC = 14

The normal blood pH range is 7.35 to 7.45. In order for normal metabolism to take place, the body must maintain this narrow range at all times. When the pH is below 7.35, the blood is said to be *acidic.*

Changes in body system functions that occur in an acidic state include a decrease in the force of cardiac contractions, a decrease in the vascular response to catecholamines and a diminished response to the effects and actions of certain medications.

When the pH is above 7.45, the blood is said to be *alkalotic.* An alkalotic state interferes with tissue oxygenation

and normal neurological and muscular functioning. Significant changes in the blood pH above 7.8 or below 6.8

will interfere with cellular functioning, and if uncorrected will lead to death.

So how is the body able to self-regulate acid-base balance in order to maintain pH within the normal range?

It is accomplished using delicate buffer mechanisms between the respiratory and renal systems.

**KEY CONCEPTS**:

* The only 2 ways an *acidotic* state can exist is from either too much pCO2 or too little HCO3.
* The only 2 ways an *alkalotic* state can exist is from either too little pCO2 or too much HCO3.

**THE RESPIRATORY (LUNGS) BUFFER RESPONSE**

A normal by-product of cellular metabolism is carbon dioxide (CO2). CO2 is carried in the blood to the lungs, where excess CO2 combines with water (H2O) to form carbonic acid (H2CO3). The blood pH will change according to the level of carbonic acid present. This triggers the lungs to either increase or decrease the rate and depth of ventilation until the appropriate amount of CO2 has been re-established. Activation of the lungs to compensate for an imbalance starts to occur within 1 to 3 minutes.

**THE METABOLIC (RENAL) BUFFER RESPONSE**

In an effort to maintain the pH of the blood within its normal range, the kidneys excrete or retain bicarbonate (HCO3-). As the blood pH decreases, the kidneys will compensate by retaining HCO3- and as the pH rises, the kidneys excrete HCO3- through the urine. Although the kidneys provide an excellent means of regulating acid-base balance, the system may take from hours to days to correct the imbalance. When the respiratory and renal systems are working together, they are able to keep the blood pH balanced by maintaining 1 part acid to 20 parts base.

**ACID BASE DISORDERS**

**Respiratory Acidosis**

Respiratory acidosis is defined as a pH less than 7.35 with a PaCO2 greater than 6kpa.

Acidosis is caused by an accumulation of CO2 which combines with water in the body to produce carbonic acid, thus, lowering the pH of the blood. Any condition that results in hypoventilation can cause respiratory acidosis. These conditions include:

* Central nervous system depression related to head injury
* Central nervous system depression related to medications such as narcotics, sedatives, or anaesthesia.
* Impaired respiratory muscle function related to spinal cord injury, neuromuscular diseases, or neuromuscular blocking drugs.
* Pulmonary disorders such as atelectasis, pneumonia, pneumothorax, pulmonary oedema, or bronchial obstruction.
* Massive pulmonary embolus.
* Hypoventilation due to pain, chest wall injury/deformity, or abdominal distension

**Signs and Symptoms of Respiratory Acidosis**

Dyspnoea

Respiratory distress

Shallow respirations

Neurological

Headache

Restlessness

Confusion

Tachycardia

Dysrhythmias

Increasing ventilation will correct respiratory acidosis. The method for achieving this will vary with the cause of hypoventilation. If the patient is unstable, manual ventilation with a bag mask is indicated until the underlying problem can be addressed. After stabilization, rapidly resolvable causes are addressed immediately. Causes that can be treated rapidly include pneumothorax, pain, and CNS depression related to medications. If the cause cannot be readily resolved, the patient may require mechanical ventilation while treatment is rendered. Although patients with hypoventilation often require supplemental oxygen, it is important to remember that oxygen alone will not correct the problem.

**Respiratory Alkalosis**

Respiratory alkalosis is defined as a pH greater than 7.45 with a PaCO2 less than 4.67kpa

Any condition that causes hyperventilation can result in respiratory alkalosis. These conditions include:

* Psychological responses, such as anxiety or fear.
* Pain
* Increased metabolic demands, such as fever, sepsis, pregnancy, or thyrotoxicosis
* Medications, such as respiratory stimulants
* Central nervous system lesions

**Signs and Symptoms of Respiratory Alkalosis**

**Neurological**

* light-headedness
* numbness and tingling
* confusion
* inability to concentrate
* blurred vision

**Cardiovascular:**

* dysrhythmias
* palpitations
* diaphoresis

**Miscellaneous:**

* dry mouth
* tetanic spasms of the arms and legs

**CLINICAL APPLICATION:**

If the CO2 becomes extremely high, drowsiness and unresponsiveness may be noted.

**CLINICAL APPLICATION:**

Treatment of respiratory alkalosis centres on resolving the underlying problem.

Patients presenting with respiratory alkalosis have dramatically increased work of breathing and must be monitored closely for respiratory muscle fatigue. When the respiratory muscles become exhausted, acute respiratory failure may ensue.

**Metabolic Acidosis**

Metabolic acidosis is defined as a bicarbonate level of less than 22 mEq/L with a pH of less than 7.35. Metabolic acidosis is caused by either a deficit of base in the bloodstream or an excess of acids, other than CO2. Diarrhoea and intestinal fistulas may cause decreased levels of base.

Causes of increased acids include:

* Renal failure
* Diabetic ketoacidosis
* Anaerobic metabolism
* Starvation
* Salicylate intoxication

**Signs and Symptoms of Metabolic Acidosis**

* Neurological
* Headache
* Confusion
* Restlessness
* Lethargy
* Stupor or coma
* Cardiovascular dysrhythmias
* Warm, flushed skin
* Pulmonary Kussmaul’s respirations
* Gastrointestinal - nausea and vomiting

As with most acid-base imbalances, the treatment of metabolic acidosis is dependent upon the cause. The

presence of metabolic acidosis should spur a search for hypoxic tissue somewhere in the body. Hypoxemia can

lead to anaerobic metabolism system-wide, but hypoxia of any tissue bed will produce metabolic acids as a result of anaerobic metabolism even if the PaO2 is normal. The only appropriate way to treat this source of acidosis is to restore tissue perfusion to the hypoxic tissues. Other causes of metabolic acidosis should be considered after the possibility of tissue hypoxia has been addressed.

**CLINICAL APPLICATION:**

Current research has shown that the use of sodium bicarbonate is indicated only for known bicarbonate-responsive acidosis, such as that seen with renal failure. Routine use of sodium bicarbonate to treat metabolic acidosis results in subsequent metabolic alkalosis with hypernatremia and should be avoided.

**Metabolic Alkalosis**

Metabolic alkalosis is defined as a bicarbonate level greater than 26 mEq/litre with a pH greater than 7.45. Either an excess of base or a loss of acid within the body can cause metabolic alkalosis. Excess base occurs from ingestion of antacids, excess use of bicarbonate, or use of lactate in dialysis. Loss of acids can occur secondary to protracted vomiting, gastric suction, hypochloremia, excess administration of diuretics, or high levels of aldosterone.

**Signs and Symptoms of Metabolic Alkalosis**

Pulmonary:

* Respiratory depression
* Neurological:
* Dizziness
* Lethargy
* Disorientation
* Seizures
* Coma

Musculoskeletal

* Weakness
* Muscle twitching
* Muscle cramps
* Tetany

Gastrointestinal

* Nausea
* Vomiting

Metabolic alkalosis is one of the most difficult acid-base imbalances to treat. Bicarbonate excretion through the kidneys can be stimulated with drugs such as acetazolamide (Diamox®), but resolution of the imbalance will be slow. In severe cases IV administration of acids maybe used.

**CLINICAL APPLICATION:**

It is significant to note that metabolic alkalosis in hospitalized patients is usually iatrogenic in nature.

**SPECIAL CONSIDERATIONS**

Although the focus of this self-learning packet has been on interpretation of acid-base imbalances, the arterial blood gas can also be used to evaluate blood oxygenation. The component of the arterial blood gas used to evaluate this is the PaO2. Remember that the normal blood PaO2 value is 10.4- 13kpa.

**OXYHAEMOGLOBIN DISSOCIATION CURVE**

The oxyhaemoglobin dissociation curve is a tool used to show the relationship between oxygen saturation and the PaO2.The strength with which oxygen binds to the haemoglobin molecule has important clinical implications. If the oxygen binds too loosely, the haemoglobin may give up its oxygen before it reaches the tissues in need. If the oxygen binds too tightly, it may not transfer to the tissues at all. The strength of the oxygen-haemoglobin bond is graphically represented by the oxyhemoglobin dissociation curve below.

Several variables affect the affinity of the oxygen molecule to haemoglobin. Conditions that cause enhanced release of the oxygen molecule include acidosis, fever, elevated CO2 levels, and increased 2, 3-diphosphoglycerate (2, 3-DPG, a by-product of glucose metabolism). This change in affinity is called a shift to the right (C waveform). Conditions that keep the oxygen molecule tightly attached to haemoglobin include hypothermia, alkalosis, low PCO2, and decrease in 2, 3-DPG. This change is called a shift to the left (B waveform). A shift to the left has more negative implications for the patient than a shift to the right.

pH 7.29 (7.35-7.45) ACIDOSIS

pCO2 3.9(4.67-6.00kpa) ALKALOSIS

HCO3 18 (22-26) ACIDOSIS = Kidneys

The oxyhaemoglobin dissociation curve can be used to estimate the PaO2 if the oxygen saturation is known. The illustration demonstrates that if the curve is not shifted (A waveform), an oxygen saturation of 88% is equivalent to a PaO2 of about 60 mm Hg. With a left shift, the same saturation is equivalent to a much lower PaO2.

If evaluation of blood oxygenation is required, you can assess this by adding one additional step to your arterial blood gas analysis.

**Step Four**

Assess the PaO2. A value below 10.4kpa can indicate hypoxemia, depending on the age of the patient. Correction of a patient’s blood oxygenation level may be accomplished through a combination of augmenting the means of oxygen delivery and correcting existing conditions that are shifting the oxyhaemoglobin curve.

**SUMMARY**

Understanding arterial blood gases can sometimes be confusing. A logical and systematic approach using these steps makes interpretation much easier. Applying the concepts of acid base balance will help the healthcare provider follow the progress of a patient and evaluate the effectiveness of care being provided.

**GLOSSARY**

**ABG:** arterial blood gas. A test that analyses arterial blood for oxygen, carbon dioxide and bicarbonate content in addition to blood pH. Used to test the effectiveness of ventilation.

**Acidosis:** a pathologic state characterized by an increase in the concentration of hydrogen ions in the arterial blood above the normal level. May be caused by an accumulation of carbon dioxide or acidic products of metabolism or a by a decrease in the concentration of alkaline compounds.

**Alkalosis:** a state characterized by a decrease in the hydrogen ion concentration of arterial blood below normal level. The condition may be caused by an increase in the concentration of alkaline compounds, or by decrease in the concentration of acidic compounds or carbon dioxide.

**Chronic obstruction pulmonary disease (COPD):** a disease process involving chronic inflammation of the airways, including chronic bronchitis (disease in the large airways) and emphysema (disease located in smaller airways and alveolar regions). The obstruction is generally permanent and progressive over time.

**Diamox ™:** a carbonic anhydrase inhibitor that decreases H+ ion secretion and increases HCO3 excretions by the kidneys, causing a diuretic effect.

**Hyperventilation:** a state in which there is an increased amount of air entering the pulmonary alveoli (increased alveolar ventilation), resulting in reduction of carbon dioxide tension and eventually leading to alkalosis.

**Hypoventilation:** a state in which there is a reduced amount of air entering the pulmonary alveoli.

**Hypoxemia**: below-normal oxygen content in arterial blood due to deficient oxygenation of the blood and resulting in hypoxia.

**Hypoxia:** reduction of oxygen supply to tissue below physiological levels despite adequate perfusion of the tissue by blood.

**Iatrogenic:** any condition induced in a patient by the effects of medical treatment.

**Kussmaul’s respirations:** abnormal breathing pattern brought on by strenuous exercise or metabolic acidosis, and is characterized by an increased ventilatory rate, very large tidal volume, and no expiratory pause.

**Oxygen delivery system**: a device used to deliver oxygen concentrations above ambient air to the lungs through the upper airway.

**Oxygenation:** the process of supplying, treating or mixing with oxygen.

**Oxyhaemoglobin:** haemoglobin in combination with oxygen.

**Pneumothorax:** an abnormal state characterized by the presence of gas (as air) in the pleural cavity.

**Pulmonary Embolism:** the lodgement of a blood clot in the lumen of a pulmonary artery, causing a severe dysfunction in respiratory function.

**Thyrotoxicosis:** toxic condition due to hyperactivity of the thyroid gland. Symptoms include rapid heart rate, tremors, increased metabolic basal metabolism, nervous symptoms and loss of weight.

**SELF ASSESSMENT**

[**https://www.rdehospital.nhs.uk/e-learning/blood\_gasses/packagev5.swf**](https://www.rdehospital.nhs.uk/e-learning/blood_gasses/packagev5.swf)

**Follow the link for e resource learning for arterial blood gas sampling:**

1. The solution that would be most alkalotic would be the one with a pH of:

A. Four

B. Seven

C. Nine

D. Fourteen

2. The normal pH range for blood is:

A. 7.00 – 7.25

B. 7.30 – 7.40

C. 7.35 – 7.45

D. 7.45 – 7.55

3. The respiratory system compensates for changes in the pH level by responding to changes

in the levels of:

A. CO2

B. H2O

C. H2CO3

D. HCO3

4. The kidneys compensate for acid-base imbalances by excreting or retaining:

A. Hydrogen ions

B. Carbonic acid

C. Sodium Bicarbonate

D. Water

5. All of the following might be a cause of respiratory acidosis except:

A. Sedation

B. Head trauma

C. COPD

D. Hyperventilation

6. A patient with a prolonged episode of nausea, vomiting and diarrhoea has an ABG ordered

on admission. You might expect the results to show:

A. Metabolic acidosis

B. Metabolic alkalosis

C. Respiratory acidosis

D. Respiratory alkalosis

7. A calculated ABG value that indicates excess or insufficiency of sodium bicarbonate in the

system is:

A. HCO3

B. Base excess

C. PaO2

D. pH

8. Which of the following may be a reason to order an ABG on a patient?

A. The patient suddenly develops shortness of breath

B. An asthmatic is starting to show signs of tiring

C. A diabetic has developed Kussmaul’s respirations

D. All of the above

9. You are reviewing the results of an ABG. Both the pH and the CO2 values are abnormal

and match. The primary problem is:

A. Respiratory

B. Renal

C. Metabolic

D. Compensation

10. You are reviewing the results of an ABG. Both the pH and the HCO3 values are abnormal and match.

The primary problem is:

A. Respiratory

B. Renal

C. Metabolic

D. Compensation

11. The oxyhemoglobin dissociation curve represents the relationship between the:

A. O2 saturation and haemoglobin level

B. O2 saturation and PaO2

C. PaO2 and the HCO3

D. PaO2 and the pH

Date self-assessment completed…………………………………………….

Signature of learner…………………………………………………………….

**Procedural guideline for Arterial Puncture using the Radial Artery**

Essential Equipment

* Sterile dressing pack
* Sterile gloves
* 2% Chlorhexidine 70% Alcohol solution
* Heparinised blood gas syringe with needle
* Hypoallergenic tape
* Sharps disposal available

|  |  |
| --- | --- |
| **ACTION** | **RATIONALE** |
| 1. Where possible, explain and discuss the procedure with the patient. If the patient lacks the capacity to make decisions, the practitioner must act in the patients best interests.
 | To ensure the patient understands the procedure and gives valid, informed consent. To ensure the patients best interests are maintained (MCA, 2005) To minimise anxiety which may exacerbate symptoms. |
| 1. Check the concentration of oxygen the patient is breathing and body temperature at time of sampling.
 | Inspired oxygen concentration and temperature parameters are required to interpret ABG’s accurately. |
| 1. Check the patients current coagulation screen, platelet count, medical history and prescription chart for coagulation therapy.
 | To identify potential risk of bleeding and haematoma formation post procedure and, where appropriate to prevent puncture until coagulation is corrected. |
| 1. Prepare trolley or work space.
 | To reduce the risk of cross-infection. |
| 1. Wash hands using the 10 steps to effective hand hygiene using bactericidal soap and water or bactericidal alcohol hand rub.
 | To reduce the risk of cross-infection. |
| 1. Perform the Allen Test
 | To confirm patency of ulnar artery circulation and assess collateral circulation to the hand in the event of radial artery damage such as thrombosis. |
| 1. Locate and palpate the radial artery with the middle and index fingers of the non-dominant hand.
 | To assess maximum pulsation to ensure radial artery optimum site for successful puncture. The dominant hand will be used to perform the puncture. |
| 1. Inspect and assess the surrounding tissues and anatomical structures.
 | Check and assess surrounding tissues for excoriation or infection, poor perfusion and other puncture sites. If any of these are present, the site should not be used. |
| 1. Prepare the patient position: Gently extend the wrist. Use a rolled towel or assistance if required.
 | To reduce the risk of the patient moving unexpectedly, this could result in through puncture. To aid in flexing the hand slightly to facilitate insertion. |
| 1. Open dressing pack and equipment onto it. Expel heparin solution and withdraw the plunger of the syringe 1-2mls
 | To reduce haemolysis. Arterial pressure causes a brisk pulsatile reflex of blood into the syringe (unless severely hypotensive) To maintain the correct blood/heparin ratio. |
| 1. Place a sterile field under the patients wrist and maintain aseptic technique throughout the procedure
 | To minimise the risk of infection |
| 1. Clean site with 2% Chlorhexidine 70% Alcohol solution.
 | To minimise the risk of infection |
| 1. *Administer subcutaneous local anaesthetic if within scope of professional practice*
 | To minimise pain during the procedure. Local vasodilation effects of the local anaesthetic may reduce vasospasm, aiding a successful puncture. |
| 1. Clean hands with bactericidal skin cleaning solution.
 | To minimise the risk of infection. |
| 1. Apply sterile gloves
 | To minimise the risk of infection and prevent contamination of the hands with blood. |
| 1. Re-identify the point of maximum pulsation, hold the position with index/middle finger.
 | To guide the position of the radial artery and aid successful puncture. |
| 1. Angle the needle at 30-45° with the bevel of the needle facing upwards just distally to the finger position. Advance the needle slowly into the radial artery until a flashing pulsation is seen in the hub of the needle.
 | To minimise trauma to the artery. Rapid insertion may result in a through puncture.Pulsatile flow indicates access to the radial artery.Arterial pressure causes blood to pulsate spontaneously back into the syringe. |
| 1. Slowly aspirate by gently pulling the plunger of the syringe to a minimum of 0.6mls of blood for the sample. Ideally 2mls of blood should be collected. Note that samples of less than 1ml should be run as a capillary sample.
 | To minimise vasospasm. To minimise haemolysis.To ensure optimum volume is obtained in order to ensure an appropriate mix of heparin and blood. |
| 1. Withdraw the needle, immediately followed by application of pressure using a low-lint dressing. Return the wrist to the neutral position.
 | To prevent haematoma formation and excessive bleeding.Prolonged hyperextension may be associated with changes to median nerve conduction. |
| 1. Apply pressure for a minimum of 5 minutes or until no signs of bleeding are observed. Ask for assistance if required.
 | To minimize blood loss and to ensure pressure is exerted to prevent haematoma formation and blood loss. Assistance with pressure application may expedite sample analysis.  |
| 1. Dispose of equipment safely.
 | To prevent injury or contamination of others. |
| 1. Expel any air bubbles from the syringe and cap the syringe. Invert the syringe gently at least 15 times. Label sample according to local policy.
 | To keep sample airtight and to ensure thorough mixing of blood and heparin. Prevention of haemolysis. |
| 1. Check puncture site and apply a clean, sterile, low linting gauze dressing using ANTT. Secure with tape.
 | To maintain pressure and prevent haematoma formation. |
| 1. Clearly document procedure and rationale in the patients notes and verbally communicate arterial analysis findings to relevant clinical teams.
 | To provide an accurate record, to reduce the risk of duplication of treatment, to provide a point of reference or comparison in the event of later questions, to acknowledge accountability for your actions, to facilitate communication and continuity of care. |

(NMC, 2015; GMC, 2013; Royal Marsden Hospital Manual of Clinical Procedures, 2011; Critical Care Manual of Clinical Procedures and Competencies, 2013.)

**COMPETENCY ASSESSMENT – ABG SAMPLING**

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| --- | --- | --- | --- |
| **Subject** | **Pass****Y/N** | **Action plan****Y/N** | **Signature &****Date** |
| * 1. **Demonstrate an understanding of the practitioner’s role and responsibility in arterial blood gas sampling**

The practitioner will be expected to :-* Discuss the need for arterial blood gas sampling
* Discuss the parameters of arterial blood gases
 | 1.2.­­­­­­­­­­3. | ­­­­­­­­­­ |  |
| * 1. **Demonstrate an understanding of the anatomy and physiology of the major arteries and surrounding tissues, including veins and nerves**

The practitioner will be expected to :-* Discuss the normal anatomy and physiology of major arteries and surrounding tissues, including veins and nerves
 | 1.2.­­­­­­­­­­3. | ­­­­­­­­­­ |  |
| * 1. **Demonstrate the ability to ensure safe practice in the undertaking of arterial blood gas sampling**

The practitioner will be directly observed to determine :-* Their preparation of the patient
* The selection of the appropriate artery
* The arterial blood gas sampling
* Preparation of the sample for sampling and/or transfer to the laboratory
* The disposal of equipment
* Documentation of the procedure and the results recorded in the medical notes
* Discuss action required depending on results
 | 1.2.­­­­­­­­­­3.45.6. | **­­­­­­­­­­** |  |
| * 1. **Demonstrate an understanding of the contra-indications for performing arterial blood gas sampling**The practitioner will be expected to :-
* Discuss 6 contra-indications for performing arterial blood gas sampling
 | 1.2.­­­­­­­­­­3. | ­­­­­­­­­­ |  |

**ARTERIAL BLOOD GAS COMPETENCY PACK**

**ADDITIONAL ACTION PLANNING**

This document is to be completed as required to set SMART objectives for the learner who requires additional support to achieve their ABG competence for example further theoretical training or extra supervised sessions. To be used in conjunction with the competency assessment.

|  |  |  |
| --- | --- | --- |
| **Subject Number** | **Areas for additional action planning** | **Achieved Y/N****Refer to competency assessment**  |
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**ASSESSMENT FOR PRACTITIONERS TRAINED IN THE SAMPLING**

**OF ARTERIAL BLOOD GASSES**

Date assessment started:………….…………………………………………………………….

Date assessment completed: ………………………………………………………………

Date completed theory……………………………………………………………………………

Theory training provided by………………………………………………………………………

(Registrar level or above)

Name of Trainee (Please Print): ………………………………………………………………

Signature of Trainee: ………………………………………………………………

Name of Assessor (Please Print): ………………………………………………………………

Signature of Assessor: ………………………………………………………………

Update of competencies required………………………………………………………………

NOTES