# **Workshop: Blood Gases and acid-base data**

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## LEARNING OBJECTIVES

1. Describe the qualitative changes in arterial blood pH. PCO2 and Base Excess in the following acid-base disturbances:

(i) Acute respiratory acidosis

(ii) Acute respiratory alkalosis

1. For (i) and (ii) above, describe the qualitative changes in arterial blood pH, PCO2 and Base Excess following renal compensation.
2. Describe the qualitative changes in arterial blood pH. PCO2 and Base Excess in the following acid-base disturbances:

(i) Metabolic acidosis with respiratory compensation

(ii) Metabolic alkalosis with respiratory compensation

Comment on the mechanism whereby metabolic changes in acid-base status lead to alteration in ventilation and hence respiratory compensation.

1. Describe the qualitative changes in arterial blood pH. PCO2, Base Excess and PO2 in a patient with (i) Type I respiratory failure (ii) Type II respiratory failure, in each case after full renal compensation.

### INTRODUCTION

### The overall aim of this session is to introduce you to the principles underlying the interpretation of blood gas data and to consider these in the context of the problems that you will meet in clinical practice as you arrive on the wards. The topic is a good example of some applied respiratory and renal physiology.

### STRUCTURE OF SESSION

There will be a short introductory lecture (some essential graphs for this are included in this handout). You will then have about 45 minutes to work through the self-directed learning exercise on this topic. There is no designated space in which to do this but you can remain in the LT or go and sit in the café or surrounding area. You can work alone or discuss the problems with other students (or a bit of both). I will be available in the LT1 to discuss any problems you might have. Try to understand the examples as you go through and then attempt the problems at the end. Please return to the LT1 at the designated time to go through the exercises.

**SELF DIRECTED LEARNING EXERCISE/GROUP DISCUSSION**

On the hospital wards, in the operating theatre and in the Intensive Care Unit you will be presented with a print out from a blood gas analysis upon which you will be required to assess the ventilatory and acid-base status of the patient. The analysis will have measured and computed the following on a sample of arterial blood taken from the patient. (The interpretation of the data requires you to know whether the patient was breathing air or air enriched with oxygen)

**Normal range of values:**

Measured Hb 13.3 - 17.7 g/dl

 pH 7.37 - 7.45 units

 Pco2 4.7 - 6.4 kPa (35 - 48 mm Hg)

 Po2 Over 10.7 kPa (80 mmHg)

Calculated Base excess -2 - +2 mmol/l.

Firstly, let us consider the acid base status of the patient. This is determined by a complex balance between acid and base input to the patient, acid or base loss from the patient (via the lungs and/or kidneys) and the products of metabolism. The changes in the arterial blood reflect this complex interaction.



**Figure 1.**

Curved relationship between arterial PCO2 (kPa or mm Hg) and alveolar ventilation

(litres/min) at three different levels of CO2 production (dashed, continous and dash dot curves) in ml/min. Changers in CO2 production produce changes in alveolar ventilation such that PCO2 remains within the normal range, between the continous vertical lines at PCO2 4.7 and 6.4 kPa or between 35 and 48 mmHg. Thus a normal PCO2 implies a normal alveolar ventilation and chemical control of CO2 whatever the CO2 production. Thus a rise in CO2 production from 200 to 300 ml/min leads to an increase of alveolar ventilation from approximately 4 to 6 litres/min

**Acute respiratory acidosis\* and alkalosis\* (uncompensated)**

(\* sometimes the terms acidaemia and alkalaemia are used, especially to describe situations where the arterial blood pH is outside the normal range).

These are the simplest to explain.

###### **Example 1**

A patient is brought into the casualty department semi-conscious. The patient was found at home with an empty bottle of vodka and an empty bottle of sleeping pills nearby. The patient was not rousable but responded to painful stimuli. Blood gas analysis showed:

pH = 7.16

 Pco2 = 10.7 kPa (80 mmHg)

 Po2 = 5.3 kPa (40 mmHg)

 Base excess = +1.0 mmol/l.

 **Diagnosis: Acute respiratory acidosis**.

In other words, this patient is not breathing enough as a result of drug-induced central neural respiratory depression. The Pco2 is therefore raised and because CO2 (in H2O) is acidic, the pH is correspondingly reduced. We know that the fall in pH is completely explained by the rise in Pco2 because the base excess is in the normal range. (This is explained in detail below).

###### **Example 2**

A patient suffered a Catastrophic stroke and following this event respiration was seen to be irregular and inadequate (rise in arterial Pco2 recorded). The patient was intubated and ventilated with an inspired oxygen concentration of 40%. Analysis of a blood sample 4 hours later showed:

pH = 7.63

 Pco2 = 2.7 kPa (20 mmHg)

 Po2 = 35.3 kPa (265 mmHg)

 Base excess = -1.2 mmol/l.

 **Diagnosis: Acute respiratory alkalosis**

Here, the acid base situation is opposite to that in Example1. Although this patient also had ventilatory insufficiency (due to stroke), the subsequent medical intervention resulted in a state of overventilation causing the Pco2 to fall and the pH to rise. In this case, the high Po2 results from the fact that the patient is being given additional oxygen to breathe.

**Metabolic acid-base disturbances and the role of the kidney**

The disturbances described above are due to alterations in the gaseous acid, carbon dioxide. Now we must consider changes due to metabolic acids (e.g. lactic acid, ketoacids, sulphuric acid) and the effect of acid excretion by the kidneys.

So far we have ignored the effect of changes in bicarbonate concentration ([HCO3-]) in the acid base status of the blood. [HCO3-] is one of the two variables that determine the [H+] or pH of the blood.

Since CO2 + H20 ⇔ H2CO­3 ⇔ H+ + HCO3-

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[H+] = K [H2CO3] pH = pK + log [HCO3-]

 [HCO3-] [H2CO3]

A knowledge of the [HCO3-] is currently required for the complete interpretation of the acid base status of the blood. The problem is that [HCO3-] in the blood is determined by several different factors which makes it difficult for the student to understand. The base excess was designed (see later) to aid interpretation.

The factors which affect the [HCO3-] within the blood are:

**(a)** Gaseous (due to carbon dioxide)

 [HCO3-] rises and falls directly with the Pco2

**(see Fig 2)**

**(b)** Metabolic (non gaseous)

 [HCO3-] falls when metabolic acids are 'buffered' in the blood.

 e.g. HLa (Lactic acid) + NaHCO3 NaLa (lactate) + H2CO3

**(see Fig. 3)**

the HCO3- lost in this buffering is normally “regenerated” in the kidney in conjunction with the excretion of H+ ions.

 [HCO3-] rises if sodium bicarbonate is administered orally or intravenously.

**(c)** Renal

[HCO3-] rises when acid excretion by the kidney is increased and falls when there is a reduction in, or failure of excretion of acid by the kidney.

**(see Fig. 3)**

# **Base Excess**

The purpose of deriving a figure called base excess is to enable you to determine how much of a disturbance of the acid base status of the blood (i.e. change in [H+] is due to (a) changes in the production or ingestion of metabolic acids and bases and (b) due to changes in the excretion of acid by the kidney. A computer in the blood gas analyser calculates a theoretical [HCO3-] based on the patient’s measured Pco2 assuming no metabolic or renal disturbance. The actual [HCO3-] is then calculated from the patient’s measured pH and Pco2. The difference between actual and theoretical [HCO3-] is the base excess in mmol/l. Changes in [HCO3-] due to alterations in Pco2 are therefore eliminated and if a change is present it is solely due to a metabolic acid base disturbance or a change in the renal excretion of acid. The Pco2 can be above or below normal but the base excess will be close to zero providing there is no metabolic acid base disturbance and no change in the renal excretion of acid.

The normal range for base excess is +2 to -2.

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**Figure 2.** The CO2 produced in the tissues is buffered by Hb in the red cell. Carbonic acid is formed with water which dissociates into hydrogen and bicarbonate ions. CO2 also combines with Hb to form carbamino CO2 and an hydrogen iron. The hydrogen ions are buffered by Hb. The reverse process occurs in the lungs.

Renal compensation



**Figure 3.**

Metabolic acids (sulphuric, lactic etc) are first buffered by bicarbonate.

HLA + NaHCO3 NaLA+H2C03

H2C03 is excreted in the lungs as C02 and H20. Thus, as a result of the buffering there is a loss of bicarbonate. This is regenerated in the kidney. CO2 produced in the kidneys form carbonic acid which dissociates into an hydrogen and bicarbonate ion. Hydrogen ions are transported into the glomerular filtrate and bicarbonate into the ECF and blood

A rise in base excess is due to an increase in renal excretion of acid or ingestion or the administration of base. It may also be due to loss of acid from vomiting. The result is a metabolic alkalosis.

A fall in base excess is due to the overproduction of metabolic acids (e.g. lactic acid) or the ingestion of acid. It may also be due to a reduction in, or failure of, acid excretion by the kidney or to excessive loss of alkali from the intestine with diarrhoea. The result is a metabolic acidosis.

###### **Example 3**

A patient with abdominal pain due to a duodenal ulcer was admitted to the medical ward with persistent vomiting (loss of HCl). He was also taking large quantities of sodium bicarbonate to ease the pain. A sample of arterial blood revealed:

 pH 7.54

 Pco2 6.7 kPa (50 mmHg)

 Po2 11.1 kPa (83 mmHg)

 Base excess + 17 mmol/l.

**Diagnosis: Metabolic alkalosis with respiratory compensation.**

The pH would have been more alkaline (7.63) if there had not been a fall in alveolar ventilation and rise in Pco2.

**Example 4**

A 52 year old man was admitted unconscious to casualty. He was a known diabetic on daily insulin. One week ago he had developed a chest infection. He stayed at home and because he stopped eating he stopped his insulin. Over the preceding 2 days he had become increasingly drowsy and in the morning of admission he was unrousable. The arterial blood gas results were:

 pH = 7.19

 Pco2 = 4.0 kPa (30 mmHg)

 Po2 = 13.3 kPa (100 mmHg)

 Base excess - 16.5 mmol/l.

 **Diagnosis: Metabolic acidosis** **with respiratory compensation.**

The pH would have been more acid (7.13) if the acidosis had not stimulated alveolar ventilation leading to a fall in Pco2.

**Chronic respiratory acidosis and alkalosis (compensated)**

In some clinical situations, a patient’s ventilation may be suppressed or stimulated over a period of days, weeks or even years.

# **Example 5.**

# During extremely cold weather, a 68 year old woman with chronic obstructive pulmonary disease was discovered at home in a semi-conscious state. On admission to hospital her blood gas data were as follows:

 pH = 7.36

 Pco2 = 8.0 kPa (60 mmHg)

 Po2 = 5.3 kPa (40 mmHg)

 Base excess = +7 mmol/l.

## Diagnosis: Respiratory acidosis with renal compensation

# Because of the nature of this patient’s condition, it is likely that chronic hypoventilation has existed for some time. In this situation, the chronic respiratory acidosis will result in additional loss of acid by the kidney as a compensatory response to maintain pH homeostasis.

# **Example 6**

# A 36 year old man presents himself at Casualty in a distressed state. He reports episodic chest pain over the past 2 weeks with very little sleep. He gives a family history of ischaemic heart disease. His blood gas findings were as follows:

 pH = 7.46

 Pco2 = 3.3 kPa (25 mmHg)

 Po2 = 15.3 (115 mmHg)

 Base excess = -5 mmol/l.

# Further investigations revealed that his chest pain was probably due to muscle strain and that his perceived heart problem had resulted in considerable anxiety.

## Diagnosis: Respiratory alkalosis with renal compensation

Other examples for you to work out (all breathing air)

1.

pH = 7.25

Pco2 = 10.7 kPa (80mmHg)

Po2 = 6.7 kPa (50 mmHg)

[HCO3] = 35 (mmol/L)

Base excess = + 4 mmol/L

**Diagnosis?**

3.

pH = 7.40

Pco2 = 4 kPa (30 mmHg)

Po2 = 13.3 kPa (100 mmHg)

[HCO3] = 18 (mmol/L)

Base excess = -5 mmol/L

**Diagnosis?**

5.

pH = 7.20

Pco2 = 3.3 kPa (25 mmHg)

Po2 = 14.7 kPa (110 mmHg)

[HCO3] = 10 (mmol/L)

Base excess = -18 mmol/L

**Diagnosis?**

7.

Patient age 50 admitted to hospital

with pneumonia

pH = 7.64

Pco2 = 4.3 kPa (32 mmHg)

Po2 = 10.0 kPa (75 mmHg)

[HCO3] = 33 (mmol/L)

Base excess = + 17 mmol/L

**Diagnosis?**

2.

pH = 7.50

Pco2 = 2.7 kPa (20 mmHg)

Po2 = 14.7 kPa (110 mmHg)

[HCO3] = 15 (mmol/L)

Base excess = -5 mmol/L

**Diagnosis?**

4.

pH – 7.19

Pco2 = 8 kPa (60 mmHg)

Po2 = 6.7 kPa (50 mmHg)

[HCO3] = 22 (mmol/L)

Base excess = - 7 mmol/L

**Diagnosis?**

6.

pH = 7.55

Pco2 = 6.7 kPa (50 mmHg)

Po2 = 9.3 kPa (70 mmHg)

[HCO3] = 44 (mmol/L)

Base excess = +18 mmol/L

**Diagnosis?**

8.

Two patients with identical blood

gases

pH = 7.21

Pco2 = 9.3 kPa (70 mmHg)

Po2 = 10.7 kPa (80 mmHg)

[HCO3] = 33 (mmol/L)

Base excess = +2.2 mmol/L

**Diagnosis?**

Having looked at acid-base status, we now need to consider, how blood gas findings additionally inform us about the patient’s ventilatory function. We have already considered the effect that alveolar hypoventilation and hyperventilation have on arterial Pco2. However, if the patient is breathing air, inspection of the arterial Po2 will provide important information about the patient’s lung disease.

Look again at Example 1. The high Pco2 indicates inadequate alveolar ventilation and as a result of this the arterial Po2 is correspondingly reduced. This is referred to as **Type II Respiratory Failure**.

**Example 7**

A patient with COPD is admitted to hospital with severe breathlessness and confusion after developing a chest infection. Blood gas analysis reveals the following:

 pH = 7.39

 Pco2 = 5.6 kPa (42 mmHg)

 Po2 = 4.7 (35 mmHg)

 Base excess = 0 mmol/l.

In this case, the overall alveolar ventilation is adequate (because Pco2 is normal). However, the low Po2 (arterial hypoxaemia) indicates that despite this, the lungs are unable to adequately oxygenate the blood. This is accounted for by a mismatching of the **ventilation** and the **perfusion** (blood flow) to the lungs. This is a common problem in respiratory disease and is referred to as **Type I Respiratory Failure**.

(see Fig. 3)

**Figure 4:** The continuous oblique line defines the normal relationship between arterial PO2 and PCO2. Thus if PCO2 goes up with alveolar hypoventilation, then PO2 goes down (and viva versa with alveolar hyperventilation). Because the oblique line is linear, addition of the PO2 and PCO2 will give the same value at any point on the line (approximately 16 Kpa). This relationship is very useful in characterising Type 1 from Type II respiratory failure.

**Examples**: A:Type II failure (PCO2 10 KPa, PO2, 7.5 KPa)

 B: Type I failure (PCO2 3.5 KPa, PO2, 8.0 KPa)

 C: Combined Type I&II failure (PCO2 7.5 KPa, PO2, 5.0 KPa)