ABG interpretation

Introduction

Interpreting an arterial blood gas (ABG) is a crucial skill in acute medicine. It is especially important in critically ill patients. Its worth emphasizing that the interpretation begins with understanding clinical history.

----- XXXX Diagnostics ------

Blood	Gas	Report
248	05:36	Jul 22 2000
Pt ID	2570 / 00	
Measured	**37.0° C**	
pH	7.463	
pCO_2	44.4	mm Hg
pO_2	113.2	mm Hg
Corrected	**38.6° C**	
pH	7.439	
pCO_2	47.6	mm Hg
pO_2	123.5	mm Hg
Calculated Data		
HCO_3 act	31.1	mmol / L
HCO_3 std	30.5	mmol / L
BE	6.6	mmol / L
O_2 CT	14.7	mL / dl
O_2 Sat	98.3	%
ct CO_2	32.4	mmol / L
pO_2 (A - a)	32.2	mm Hg
pO_2 (a / A)	0.79	
Entered Data		
Temp	38.6	ºC
ct Hb	10.5	g/dl
FiO_2	30.0	%

Now, check the patient identification data in the report.

Note down the details in "entered data" like temperature, hemoglobin and FiO2. It is a good habit to note down PEEP value at the time of samling, if the patient is on mechancial ventilation.

The measured values are pH, pCO2 and pO2. Most ABG reports will provide the normal range of each data with corresponding unit.

Let us see how the machine calculates the parameters from known values of pH, P CO2 and manually entered values of temp and FiO2.

HCO3 is obtained from Henderson-Hasselbach equation:

This is actual HCO3. Standard Bicarbonate is plasma HCO3 after equilibration to a PCO2 of 40 mm Hg.

Base Excess: is calculated using Sigaard-Andersen equation.

TCO2 (total CO2) is a measure of carbon dioxide which exists in several states: CO2 in physical solution or loosely bound to proteins, HCO3 and carbonic acid (H2CO3).

It is either measured on plasma by automated chemistry analyzers or is calculated from pH and PCO2 measured on whole blood gas analyzers.

When a cartridge includes sensors for sodium, potassium, chloride, pH and PCO2, anion gap can be calculated.

Anion gap is reported as the difference between the commonly measured cations sodium and potassium and the commonly measured anions chloride and bicarbonate. The size of the gap reflects unmeasured cations and anions and is therefore an analytical gap.

ABG also analyzes gas exchange. Remember, PaO2 is directly measured by a Clark electrode.

The A-a gradient, or the alveolar-arterial gradient, measures the difference between the oxygen concentration in the alveoli and arterial system.

Alveolar oxygen (PAO2) is influenced by the FiO2, barometric pressure (high altitude), PaCO2 increase (respiratory depression)

Thus, PA O2 =(Pb-PH2O) x FiO2 – (PaCO2/0.8)

We have obtained the values of FiO2 and PCO2.

Pb is the total atmospheric pressure, which is 760mmHg at sea level, and we reduce P H_2O, which is = 47 mmHg assuming 100% humidity in the alveoli.

Substituting the values in the formula; we arrive at a simplified equation for alveolar PO_2 at sea level and on room air;

$PA\ O_2 = 150 - (PaCO_2 \times 1.25)$

If FiO_2 is known, we may put it as, $PAO_2 = 710 \times FiO_2 - (PaCO_2 \times 1.25)$

Let us take FiO_2 as 0.40 and $PaCO_2$ as 40mmhg, substituting in the equation, alveolar PO_2 comes to 234 mmHg

Now look at the ABG report for arterial PO_2; which is, say, 228. Now the A-a gradient is 234-228; 6 mmHg.

A normal A–a gradient for a young adult non-smoker breathing air, is between 5–10 mmHg. Normally, the A–a gradient increases with age.

A conservative estimate of normal A–a gradient is less than

[age in years/4] + 4.

Thus, a 40-year-old should have an A–a gradient less than 14 mmHg.

Normal A-a gradient

Alveolar hypoventilation (elevated $PACO_2$)

Low PiO_2 ($FiO_2 < 0.21$ or barometric pressure < 760 mmHg)

Raised A-a gradient

Diffusion defect (rare)

V/Q mismatch

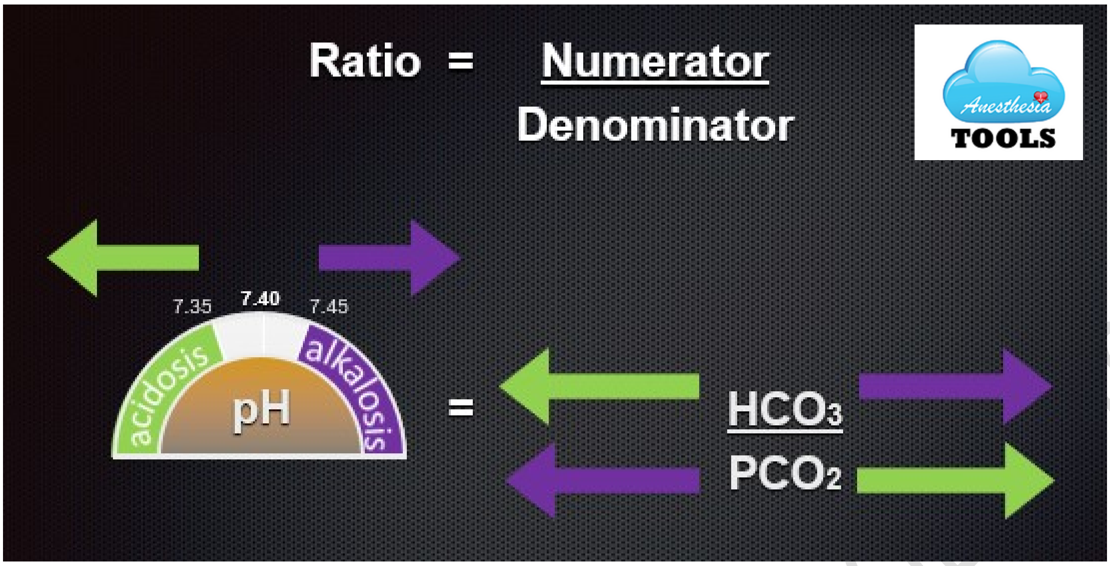
Right-to-Left shunt (intrapulmonary or cardiac)

Increased O_2 extraction (CaO_2-CvO_2)

Most blood gas machines estimate saturation from idealized oxygen dissociation curve.

Gold standard is co-oximetry.

Step-by-step interpretation



When pH decreases, its acidemia. It can be due to increase in PCO2 or decrease in HCO3. Since changes in PCO2 occurs rapidly, we give preference to PCO2 first.

Similarly, when pH increases, it leads to alkalemia. This happens with reduction in PCO2 or rise in HCO3.

Step 1: If pH < 7.35, it is acidemia; if pH >7.45, it is alkalemia.

Step 2: Find out which is the primary disturbance. If the change in pH is in tune with the change in pCO2 the primary disturbance is respiratory. (eg, pH is <7.35 and pCO2>45mmHg, it is primary respiratory acidosis). Otherwise, check the trend in HCO3, to diagnose a primary metabolic change. (eg, if pH <7.35, pCO2 <40mmHg, HCO3 <22, it is primary metabolic acidosis).

Step 3: If there is a primary respiratory disturbance, is it acute?

Expect pH drop by 0.08 for every 10mm rise in PCO2 (acute)

Expect pH drop only by 0.03 for every 10mm rise in PCO2 (chronic)

Step 4

For a respiratory disorder, check whether renal/metabolic compensation OK.

Remember the rules of thumb discussed in the previous module.

“One for 10” (acute respiratory acidosis)

“Four for 10” (chronic respiratory acidosis)

“Two for 10” (acute respiratory alkalosis)

“Five for 10” (chronic respiratory alkalosis)

Step 5 If the disturbance is metabolic is the respiratory compensation appropriate?

Again, the rest of our rules of thumb. One and a half plus 8 for met acidosis and point seven plus 20 for met alkalosis.

Expect PCO2 = (1.5 x [HCO3]) + 8 ± 2 (Metabolic Acidosis)

Expect PCO2 = (0.7 x [HCO3]) + 20 ± 5 (Metabolic Alkalosis)

Step 6 If there is metabolic acidosis, calculate anion gap?

Anion Gap = (Na + K) – (Cl + HCO3) usually <12

We can differentiate between normal anion gap acidosis and high anion gap acidosis.

Step 7 Does the anion gap explain the change in bicarbonate?

Δ anion gap is given by (Anion gap -12) ~ Δ [HCO3]

If Δ anion gap is greater; consider additional metabolic alkalosis

If Δ anion gap is less; consider a non-anion gap metabolic acidosis (NAGMA)

Metabolic acidosis with NORMAL anion gap

Renal Tubular Acidosis

Diarrhea

Uretero-enteric fistula

Hyperalimentation - TPN

Acetazolamide and other Carbonic anhydrase inhibitors

Metabolic acidosis with raised anion gap

Methanol

Uraemia

Diabetic ketoacidosis (and alcoholic/starvation ketoacidosis)

Propylene glycol

Isoniazid

Lactate

Ethylene glycol

Salicylates

Delta gap

Delta Gap = (Actual AG - 12) + HCO3

Normal range is 18-30.

If delta gap is less than 18, consider additional normal gap metabolic acidosis.

If delta gap is more than 30, consider additional metabolic alkalosis.

Practice problem 1:

60 year old male smoker with progressive respiratory distress and somnolence.

----- XXXX Diagnostics ------

Blood Gas Report

248	05:36	Jul 22 2019
Pt ID	2570 / 00	
Measured		37.0°C
pH		7.301
pCO_2		76.2 mmHg
pO_2		45.5 mmHg
Calculated		Data
$HCO_{3\ act}$		36.1 mmol/L
O_2 Sat		78%
pO_2 (A - a)		9.5 mmHg
pO_2 (a / A)		0.83
Entered Data		
FiO_2		21%

Step 1: pH< 7.35 =Acidemic

Step 2: pCO2>45mmg, accounts for acidemia = primary respiratory acidosis

Step 3: To find out if acute/chronic

As per our formula, if it is an acute resp acidosis, expected pH should be 7.11, But the given pH is 7.3

Δ PCO2 = 76 - 40 = 36

A/c Δ pH = 36/10 x0.08 = 0.29

Exp pH = 7.40 - 0.29 = 7.11

C/c Δ pH = 36/10 x0.03 = 0.10

Exp pH = 7.40-0.10 = 7.30

Moving ahead, calculate for chronic disorder, and pH comes to 7.3.

Thus, we conclude, this is chronic respiratory acidosis.

Step 4: to check extent of metabolic compensation.

Applying our rule of thumb, “four for 10”, rise in PCO2 is 36.

So, rise in HCO3 should be 4 multiplied by 36/10…. it comes to 14.4

Therefore, expected HCO3 will be 24+14.4 = 38.4

Look at the HCO3 in the report, it is 36.1, which is almost close to our calculated value

Now we can add partial metabolic compensation to our diagnosis.

Further, check for gas exchange.

Here, pO2 is very much below normal, indicating hypoxemia.

Have a look at A-a gradient… it is given directly, so no need to calculate ourselves. Here the value is in the normal range. So, hypoxemia is due to hypoventilation.

The decreased PO2 with elevated PCO2 is classical of Type 2 respiratory failure.

Now we have a detailed picture and it correlates with the clinical presentation.

Practice problem 2:

A 17-year-old girl presents to A&E complaining of a tight feeling in the chest, shortness of breath, some tingling in the fingers and around the mouth. She has no significant past medical history and are not on any regular medication.

----- XXXX Diagnostics ------

Blood Gas Report

256	05:36	Apr 02 2019
Pt ID	2574 / 02	
Measured		37.0°C
pH		7.49
pCO_2		24 mmHg
pO_2		105 mmHg
Calculated		Data
HCO_3 act		22 mmol/L
BE		+2
O_2 Sat		98%
Entered Data		
FiO_2		21%

Key:

Step 1: Look at the pH, it is more than 7.45—-so, it is alkalemia.

Next, look at the PCO2. Low PCO2 goes with alkalemia;

Check HCO3, not much change.

Now, we can put it as primary res alkalosis.

Next, we explore compensation, here we look at metabolic component or HCO3

It should decrease by our rule of thumb, 2 for 10.

here drop in PCO2 is 16.

Hence drop in HCO3 should be 2 times 1.6; that is 3.2; which is yet to happen in our ABG report. May be it will take its own time to drop HCO3 and bring pH toward normal.

Regarding oxygenation, PO2 is normal, rather no hypoxia.

Practice problem 3:

A 35-year-old woman with type 1 diabetes is brought to the emergency department by ambulance after being found severely unwell in her house. She has not been eating for the past few days due to a vomiting illness and, as a precaution, has also been omitting her insulin.

She appears drowsy and peripherally shutdown, with very dry mucous membranes. Her breath smells of acetone and her respirations are deep and sighing.

Glucometer - 450mg%

----- XXXX Diagnostics ------

Blood Gas Report

916	18:36	Jan 26 2008
Pt ID	006545	

Measured	37.0°C
pH	7.05
pCO_2	11 mmHg
pO_2	187 mmHg
Calculated	Data
HCO_3 act	6 mmol/L
BE	-25.2
O_2 Sat	99.8%

Key:

Step 1: pH shows severe acidemia.

Next, we check if PCO2 can account for acidemia. It's very much decreased, so it is not the primary disorder.

Now let's check HCO3…HCO3 value of 6 very much goes with the severe acidemia.

In addition, a huge negative value BE also supporting our conclusion.

So, we are dealing with severe metabolic acidosis.

In case of metabolic acidosis, we need to calculate Anion gap.

Here's the remaining lab reports.

Lab reports:

Lactate	1 mmol/L
K	4.6 mmol/L
Na	141 mmol/L
Cl	96 mmol/L
iCa+	1.25 mmol/L
Hb	12 g/dL
Glucose	450 mg/dl

Now we can calculate anion gap, which comes to 43.6.

This means that there are huge amounts of unmeasured anions, like say, ketone bodies in this case.

Now we refine our conclusion as High anion gap severe met acidosis.

Practice problem 4:

A 21-year-old woman presents to the emergency department with a 6-h history of worsening breathlessness and wheeze. She has a history of asthma, with two previous exacerbations requiring hospital admission. She now feels very breathless and is obtaining no relief from her salbutamol inhaler.

Pulse 115 beats/min; RR 30bpm
Blood pressure 120/80 mmHg

SpO2 96% (room air)
Peak expiratory flow 160 L/s (predicted = 400 L/s)

----- XXXX Diagnostics ------

Blood Gas Report

713	18:36	May 28 2014
Pt ID	00654545	
Measured		37.0° C
pH		7.38
pCO_2		43 mmHg
pO_2		76 mmHg
Calculated		Data
HCO_3 act		24mmol/L
BE		-1.3
O_2 Sat		96%
Entered Data		
FiO_2		21%

Key:

pH is in normal range.

pCO2 is acceptable

HCO3 is normal.

Patients with acute exacerbations of asthma # should have a low Paco2 due to the increased respiratory rate and effort (↑alveolar ventilation). A PCo2 of 43 mmHg suggests that the patient is struggling to overcome the obstruction to airflow and, perhaps, beginning to tire from the effort of breathing. Consequently, her Paco2 signals a life-threatening attack.

The intensive care unit should be informed immediately of any patient with acute severe asthma and life-threatening features. Patients must receive intensive treatment and monitoring, including repeated ABG measurements to assess response and identify the need for intubation.

Practice problem 5

A 60-year-old man is brought to the Emergency Department after a witnessed out-of-hospital cardiac arrest. The paramedics arrived after 7 min, during which CPR had not been attempted. His initial rhythm was VF and the paramedics subsequently restored a spontaneous circulation after the 3rd shock.

On arrival:

Intubated, ventilated with 50% oxygen

P 120 min , BP 150/95 mmHg

Comatose (GCS 3)

----- AAAA Diagnostics ------

Blood	Gas	Report
Measured		**37.0° C**
pH	7.10	
pCO_2	47	mm Hg
pO_2	56	mm Hg
Calculated		**Data**
HCO_3 act	14	mmol / L
BE	-10	mmol / L
Entered		**Data**
FiO_2	50.0	%

Key: Severe acute metabolic acidosis with respiratory acidosis

Feedback: saneeshpj@yahoo.com

A typical ABG result after prolonged cardiac arrest. There is a mixed metabolic and respiratory acidosis – the predominant component is metabolic, with significant impairment of oxygenation.

Treatment will include:

Increase the FiO2 – this should increase the PaO2.

Increase the minute ventilation to reduce the PaCO2 – this will quickly increase the pH.
Optimise the cardiac output – increased oxygen delivery to the tissues will restore aerobic metabolism, reduce the lactic acidosis and slowly restore the pH towards normal.
Bicarbonate is not indicated as restoring cardiac output will restore plasma bicarbonate.

Practice problem 6

A 64-year-old woman is referred to critical care after becoming unwell 48 h after an invasive urology procedure.

She appears flushed and sweaty, with a pyrexia of 39.8°C. She has a sinus tachycardia of 122 beats/min and a blood pressure of 100/65 mmHg. C-reactive protein is elevated at 267 mg/dL. Her observation chart and ABG results are shown here.

Laboratory reports:

Lactate	5.1 mmol/L	(0.4–1.5)
K	4.1 mmol/L	(3.5–5)
Na	140 mmol/L	(135–145)
Cl	101 mmol/L	(95–105)
iCa^{+}	1.1 mmol/L	(1–1.25)
Hb	15.0 g/dL	(11.5–16)
Glucose	6.8 mmol/L	(3.5–5.5)

----- AAAA Diagnostics ------

Blood	Gas	Report
Measured		**37.0° C**
pH	7.36	
pCO_2	31.5	mm Hg
pO_2	203	mm Hg
Calculated		**Data**
HCO_3 act	17.3	mmol / L
BE	-6.9	mmol / L
SO_2	100	%

Key: Mild hyperventilation (secondary);

Compensated metabolic acidosis

Lactic acidosis, likely due to global tissue hypoperfusion