ABG AND ELECTROLYTE LOUNGE

DIFFERENTIAL DIAGNOSIS OF ACID BASE DISORDERS

1. High Anion Gap Metabolic Acidosis

C	Carbon Monoxide, Cyanide
A	Alcohol, Alcoholic Ketoacidosis
T	Toluene
M	Metformin, Methanol
U	Uraemia
D	Diabetic Ketoacidosis
P	Paraldehyde, Phenformin, Paracetamol, Propylene glycol
I	Iron, Isoniazid
L	Lactic acidosis (any cause)
E	Ethylene glycol
S	Salicylates

2. Causes of Lactic Acidosis

Type A: Imbalanced oxygen supply and demand	Type B: Metabolic derangements
Carbon monoxide	B2 agonists
Shock	Cancer
Severe anaemia	Cyanide
Severe hypoxia	Ethanol
Excessive oxygen demand:	Hepatic failure
Fever, seizure,	Ketoacidosis
exercise, shivering	Metformin / Phenformin
	Sepsis
	Thiamine deficiency
	Inborn errors of metabolism

3. Non-anion Gap Metabolic Acidosis

U	Ureteroenterostomy
S	Small bowel fistula
E	Extra chloride, Normal saline hydration
D	Diarrhoea
C	Carbonic anhydrase inhibitors (acetozolamide, topiramate etc)
A	Adrenal insufficiency
R	Renal tubular acidosis
P	Pancreatic fistula

4. Causes of a low anion gap

Increased cations	Calcium, magnesium, lithium, multiple myeloma
Decreased anions	Dilution, hypoalbuminaemia
Artefactual	Bromism, Iodism, Propylene glycol, Triglycerides

5. Metabolic Alkalosis

- **C** Contraction (volume contraction)
- **L** Licorice, diuretics
- **E** Endocrine (Hyperaldosteronism, Bartter's, Cushing's, Conn's)
- **V** Vomiting, NG suction (chloride loss)
- **E** Excess alkali (antacids, dialysis, milk-alkali syndrome)
- **R** Refeeding alkalosis
- **R** Renal bicarbonate retention (Hypochloraemia, Hypokalaemia, Chronic hypercapnia)

6. Respiratory acidosis

Acute

- Airway obstruction
- Aspiration
- Bronchospasm
- CNS depression
- Muscle weakness
- Pulmonary disease

Chronic

- Chronic lung disease
- Neuromuscular disorders
- Obesity

7. Respiratory alkalosis

- **C** CNS disease (Raised ICP)
- **H** Hypoxia (Altitude, anaemia, VQ mismatch)
- **A** Anxiety
- **M** Mechanical hyperventilation
- **P** Progesterone, pregnancy
- **S** Sepsis, Salicylates and other toxins (nicotine, xanthines)

ABG OF THE WEEK #01

Describe and interpret this arterial blood gas:

History: 40y male found in his garage with altered mental state and brought to ED. His arterial blood gas on arrival follows (FiO2 = 0.21%):

pH 6.80	Na 150
pCO2 21	K 4.0
pO2 110	Cl 106
HCO3 3.0	Glucose 5
BE < -30	Urea 5
COHb < 1.0%	Ethanol <0.01%
	Osmolality 334

ABG OF THE WEEK #01 - Answer

Describe and interpret this arterial blood gas:

History: 40y male found in his garage with altered mental state and brought to ED. His arterial blood gas on arrival is as follows (FiO2 = 0.21%):

pH 6.80	Na 150
pCO2 21	K 4.0
pO2 110	Cl 106
HCO3 3.0	Glucose 5
BE < -30	Urea 5
COHb < 1.0%	Ethanol <0.01%
	Osmolality 334

What is the pH? 6.80 = Severe acidaemia

What is the primary process? HCO3 3.0 = Primary metabolic acidosis

Is there compensation for metabolic acidosis?

Expected pCO2 = 1.5 x HCO3 + 8
= 1.5 x 3 + 8
= 12.5 mmHg

Measured pCO2 is higher suggesting incomplete compensation or additional respiratory acidosis

Are there other clues to diagnosis?

Anion gap = Na – (Cl + HCO3)
= 150 – (108 + 3)
= 39

Suggests elevated anion gap acidosis

Delta gap = (AG – 12) / (24 – HCO3)
= (39 – 12) / (24 – 3)
= 1.3

Consistent with elevated anion gap acidosis

Calculated osmolality = (2 x Na) + urea + glucose + ethanol
= (2 x 150) + 5 + 5 + 0
= 310 mOsmol/L

Osmolar gap = Measured osmolality – Calculated osmolality
= 334 – 310
= 24 mOsm/L
Elevated osmolar gap

Expected PAO2 = 150 – (pCO2 x 1.25)
= 150 – (21 x 1.25)
= 123.75

A-a gradient = PAO2 – PaO2
= 123 – 110
= 13

Expected A-a gradient = 40/4 + 4
= 14
Therefore normal A-a gradient

Formulation:

Description: This arterial gas shows a severe metabolic acidaemia with a grossly elevated anion gap and an elevated osmolar gap. The delta gap ratio supports a high anion gap acidosis without an additional non anion gap component. There is either incomplete respiratory compensation, or there is a coexisting process causing a respiratory acidosis. His sodium is elevated. Significantly his ethanol level is negative (meaning there is no contribution from ethanol to his elevated osmolar gap), COHb level is negative, and serum glucose is normal.

Interpretation: This severe metabolic derangement is potentially life threatening without aggressive resuscitation and specific treatment for the underlying cause. An anion gap > 30 in association with an elevated osmolar gap suggests toxic alcohol ingestion, a severe lactic acidosis (of any cause) or a ketoacidosis. Diabetic ketoacidosis is less likely in the context of normal serum glucose. Lactate should be measured and, if elevated, an underlying cause sought. Serum methanol and ethylene glycol levels can be measured if available (not readily available in Perth). Urine can be examined for calcium oxalate crystals (specific for ethylene glycol) and ketones. Suspicion of toxic alcohol ingestion would prompt loading with ethanol and transfer for urgent dialysis.

He has incomplete respiratory compensation with a normal A-a gradient; this may be due to altered mental state or respiratory depression causing hypoventilation. Care should be taken in airway management due to the risk of worsening acidosis with the loss of native respiratory compensation.

Additional information: Patient had acute renal failure with calcium oxalate crystals seen in urine. He was loaded with ethanol and dialysed and made a full recovery. He drank radiator coolant which contained 98% ethylene glycol.

ABG OF THE WEEK #02

Describe and interpret this arterial blood gas:

History: 56y female brought in by ambulance with altered conscious state after a seizure. She was unresponsive on ambulance arrival at scene, but now withdraws from painful stimulus.

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pH 7.32	Na^+ 110 mmol/L
pCO_2 31 mmHg	K^+ 4.1 mmol/L
pO_2 110 mmHg (FiO2 0.21%)	Chloride 81 mmol/L
BE -6.0	Glucose 7 mmol/L
HCO3 = 17 mmol/L	Urea 3.0 mmmol/L
Lactate 4.7	Osmolality 240 mmol/kg

ABG OF THE WEEK #02 - Answer

Describe and interpret this arterial blood gas:

History: 56y female brought in by ambulance with altered conscious state after a seizure. She was unresponsive on ambulance arrival at scene, but now withdraws from painful stimulus.

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pH 7.32	Na^+ 110 mmol/L
pCO_2 31 mmHg	K^+ 4.1 mmol/L
pO_2 110 mmHg (FiO2 0.21%)	Chloride 81 mmol/L
BE -6.0	Glucose 7 mmol/L
HCO3 = 17 mmol/L	Urea 3.0 mmmol/L
Lactate 4.7	Osmolality 240 mmol/kg

What is the pH? 7.32 = Acidaemia

What is the primary process? HCO_3 17 = Primary metabolic acidosis

Is there compensation?

Expected pCO2 = 1.5 x HCO3 + 8
= 1.5 x 17 + 8
= 33.5

Measured pCO2 (31mmHg) supports maximal respiratory compensation

Are there other clues to diagnosis?

Anion gap = Na – (Cl + HCO3)
= 110 – (81 + 17)
= 12
= Non anion gap acidosis

Calculated osmolality = (2 x Na) + urea + glucose
= (2 x 110) + 3 + 7
= 230 mmol/kg

Osmolar gap = Measured – Calculated osmolality
= 240 – 230
= 10
= Normal osmolar gap

Formulation:

Description: ABG shows a mild metabolic acidaemia which is completely compensated. There is a normal anion gap metabolic acidosis. Lactate is elevated. There is severe hyponatraemia and hypochloraemia and serum osmolality is low (normal 275 – 295 mmol/kg). Potassium, glucose and urea are normal

Interpretation: Compensated metabolic acidosis with mildly elevated lactate is consistent with the known seizure history and would be expected to improve with supportive resuscitation measures and time. Hypochloraemia is usually associated with metabolic alkalosis, so the presence of an acidosis and acidaemia in this case is a little unusual. A second process causing a metabolic acidosis is likely – the seizure and residual lactic acidosis.

Severe hyponatraemia is concerning in this setting as the cause of coma and seizures and requires rapid correction at 1-2 mmol/hr towards 120 mmol/L to prevent further seizures (monitor CNS symptoms closely) followed by slower correction not exceeding 0.5 mmol / hour once stable. Differential diagnosis of low serum osmolality in the context of severe hyponatraemia includes fluid overload / oedema states (clinically hypervolaemic), SIADH or excess hypotonic fluid consumption (normovolaemic), or excess sodium loss from renal or pre-renal sources (hypovolaemic). Differential can be narrowed by clinical assessment of volume status and measurement of urinary sodium and urinary osmolality. Other relevant investigations may include thyroid function tests, creatinine, LFT, cortisol levels, CXR, CT and a thorough medication history.

Additional information: Optimum rate and target of raising sodium in symptomatic hyponatraemia is controversial. 3% saline can be made by adding 10 ml of 20% saline to 90ml of 0.9% saline (100ml total).

ABG OF THE WEEK #03

Describe and interpret this arterial blood gas:

History: A 45y male presents by ambulance in respiratory distress. He is febrile, sweaty, pale, and drowsy, responding to painful stimuli.

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pH 7.04	Na^+ 134 mmol/L
pCO_2 106 mmHg	K^+ 4.7 mmol/L
pO_2 79 mmHg (FiO2 0.6%)	Cl^- 111 mmol/L
HCO_3^- 27 mmHg	Glucose 18.3 mmol/L
Lactate 2.1 mmol/L	Creatinine 71 mcmol/L

ABG OF THE WEEK #03 - Answer

Describe and interpret this arterial blood gas:

History: A 45y male presents by ambulance in respiratory distress. He is febrile, sweaty, pale, and drowsy, responding to painful stimuli.

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pH 7.04	Na^+ 134 mmol/L
pCO_2 106 mmHg	K^+ 4.7 mmol/L
pO_2 79 mmHg (FiO2 0.6%)	Cl^- 111 mmol/L
HCO_3^- 27 mmHg	Glucose 18.3 mmol/L
Lactate 2.1 mmol/L	Creatinine 71 mcmol/L

What is the pH? 7.04 = Acidaemia

What is the primary process? pCO_2 106 = Primary respiratory acidosis

Is there compensation?

Expected Bicarb – (Acute)
= 24 + {(Measured pCO2-40)/10}
= 24 + {(106-40)/10}
= 30.6

Actual bicarb is lower, suggesting incomplete compensation for an acute respiratory acidosis, or the co-existence of a metabolic acidosis

Are there other clues to diagnosis?

Expected PAO2 **= (713 x FiO2) – (pCO2 x 1.25)**
= (713 x 0.6) – (106 x 1.25)
= 295.3 mmHg

A-a gradient **= PAO2 – PaO2**
= 295.3 – 79
= 216.3

Expected A-a gradient **= Age/4 + 4**
= 45/4 + 4
= 15.25

Therefore there is marked hypoxia with an elevated A-a gradient

Corrected Sodium = Na + (glucose – 5)/3
= 134 + (18.3 – 5) /3
= 138.4

Anion gap = Na – (Cl + HCO3)
= 138 – (111 + 27)
= 0

Formulation:

Description: There is a severe primary respiratory acidaemia with markedly elevated pCO2 associated with hypoxia and an elevated A-a gradient suggesting V/Q mismatch. Bicarbonate is lower than expected, suggesting either incomplete compensation for an acute process, and / or a co-existing metabolic acidosis. In the presence of elevated chloride and lactate, a metabolic component is likely, however there is no anion gap when the sodium is corrected. Potassium, corrected sodium and creatinine are all within normal limits. Glucose is elevated.

Interpretation: ABG is consistent with acute, life-threatening type one and two respiratory failure, in this clinical situation probably due to an infective exacerbation of chronic obstructive pulmonary disease associated with CO2 retention. The presumed rapid rise in pCO2 is likely to have been exacerbated by the application of high flow oxygen with FiO2 0.6 (ie Non rebreather face mask). Urgent respiratory support is required, through trial of non-invasive ventilation (or intubation given low GCS) with reduction of inspired oxygen fraction to the lowest possible to maintain pO2 greater than 60 mmHg. There is a mild coexisting metabolic acidosis which would be expected to resolve with resuscitation and time. Elevated glucose may be consistent with acute stress/infection, but may also suggest previous treatment with glucocorticoids for COPD, or an underlying impaired glucose tolerance/diabetes which should be further investigated with a fasting level after resolution of the acute illness.

Additional information: Strong ion difference (Na – Cl = 27) is low (normal 38) confirms the existence of a co-existing non-anion gap metabolic acidosis. Patient was placed on bipap with rapid correction of acidosis and hypercarbia within one hour of arrival.

ABG OF THE WEEK #04

Describe and interpret this arterial blood gas:

History: 60y female presents with productive cough and increasing breathlessness.

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pH 7.37	Na 137 mmol/L
pCO2 98 mmHg	K 4.2 mmol/L
pO2 77 mmHg (FiO2 0.3)	Cl 83 mmol/L
HCO3 55 mmHg	Glucose 8.6 mmol/L
BE 24	

ABG OF THE WEEK #04 - Answer

Describe and interpret this arterial blood gas:

History: 60y female presents with productive cough and increasing breathlessness.

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pH 7.37	Na 137 mmol/L
pCO2 98 mmHg	K 4.2 mmol/L
pO2 77 mmHg (FiO2 0.3)	Cl 83 mmol/L
HCO3 55 mmHg	Glucose 8.6 mmol/L
BE 24	

What is the pH? 7.37 = normal range (tending towards acidaemia [<7.40])

What is the primary process? pCO2 98 = Primary respiratory acidosis

Is there compensation?

Expected HCO3 (chronic) **= 24 + 4 x {(Measured pCO2 - 40)/10}**
= 24 + 4 x {98 – 40)/10}
= 47.2 mmHg
= Actual pCO2 is 55 mmHg
= Fully compensated respiratory acidosis

Are there other clues to diagnosis?

Expected PAO2 **= (713 x FiO2) – (pCO2 x 1.25)**
= (713 x 0.3) – (98 x 1.25)
= 91 mmHg

A-a gradient = PAO2 – PaO2
= 91 – 77
= 14
= Normal 19 (Age/4+4)
Therefore no A-a gradient

Formulation:

Description: This ABG shows a severe, chronic respiratory acidosis which is completely compensated, with pH returning into the normal range. Actual HCO3 is slightly higher than that predicted for compensation. pO2 is below the normal range requiring oxygen supplementation, but there is no calculated A-a gradient (although very high pCO2 makes this equation less accurate). Sodium and potassium are within normal limits. Chloride is low, contributing to the metabolic alkalosis. Glucose is slightly elevated

Interpretation: Although acutely dyspnoeic, patient has a long standing, severe respiratory acidosis. ABG is close to baseline for her, given normal pH and maximal metabolic compensation. Low chloride may be due to diuretics, dehydration or chronic corticosteroid therapy (which could also contribute to elevated BSL). Care should be taken with oxygen delivery and dose to prevent worsening respiratory acidosis during the current illness.

Additional information: Strong ion difference (Na – Cl = 54) is elevated consistent with strong ion alkalosis.

ABG OF THE WEEK #05

Describe and interpret this arterial blood gas:

History: 22 year female presents feeling weak and lethargic. She has a history of an eating disorder.

pH 7.53	Na 128 mmol/L
pCO2 52 mmHg	K 2.3 mmol/L
pO2 82 mmHg (FiO2 0.21)	Cl 82 mmol/L
HCO3 43 mmHg	Glucose 5.1 mmol/L
Lactate 1.9 mmol/L	Urea 2.9 mmol/L

ABG OF THE WEEK #05 - Answer

Describe and interpret this arterial blood gas:

History: 22 year female presents feeling weak and lethargic. She has a history of an eating disorder.

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pH 7.53	Na 128 mmol/L
pCO2 52 mmHg	K 2.3 mmol/L
pO2 82 mmHg (FiO2 0.21)	Cl 82 mmol/L
HCO3 43 mmHg	Glucose 5.1 mmol/L
Lactate 1.9 mmol/L	Urea 2.9 mmol/L

What is the pH? 7.53 = Alkalaemia

What is the primary process? HCO3 43 = Primary metabolic alkalosis

Is there compensation?

Expected pCO2 **= 0.7 x HCO3 + 20 (+/-5)**
= 0.7 x 43 + 20
= 50.1 +/- 5
Actual pCO2 is 52, therefore alkalosis is fully compensated

Are there other clues to diagnosis?

Hypochloraemia (Cl 82) is consistent with metabolic alkalosis.
Sodium, potassium and urea are all low

Expected PAO2 **= 150 – (pCO2 x 1.25)**
= 150 – (52 x 1.25)
= 85 mmHg
Oxgenation is as expected with no A-a gradient

Formulation:

Description: ABG shows a fully compensated metabolic alkalosis associated with hypochloraemia, hypokalaemia and hyponatraemia. Lactate is mildly elevated, urea is slightly low. Glucose is normal. Oxygenation is adequate on air with no A-a gradient.

Interpretation: This ABG is consistent with a long standing metabolic alkalosis. In the context of an eating disorder, this pattern could be consistent with either repeated vomiting, or misuse of diuretic agents (frusemide). Differential diagnosis would include hyperaldosteronism (if associated with hypertension, although often have high-normal sodium) or severe volume depletion (from vomiting/diuretics).

Additional information: Strong ion difference (Na - Cl) elevated at 46, suggesting relative chloride debt and consistent with metabolic alkalosis.

ABG OF THE WEEK #06

Describe and interpret this arterial blood gas:

History:
A 32 year old male, a known diabetic, found unconscious at home. He was last seen 2 days ago. His arterial blood gas (FiO2 0.3) is as follows:

pH 6.72	Na 119 mmol/L
pCO2 16 mmHg	K 6.9 mmol/L
pO2 190 mmHg	Cl 98 mmol/L
HCO3 2 mmHg	Glucose 92 mmol/L
BE <-30	Urea 22 mmol/L
Osmolality 412 mmol/kg	Creat 277 umol/L

ABG OF THE WEEK #06 - Answer

Describe and interpret this arterial blood gas:

History:
A 32 year old male, a known diabetic, found unconscious at home. He was last seen 2 days ago. His arterial blood gas (FiO2 0.3) is as follows:

pH 6.72	Na 119 mmol/L
pCO2 16 mmHg	K 6.9 mmol/L
pO2 190 mmHg	Cl 98 mmol/L
HCO3 2 mmHg	Glucose 92 mmol/L
BE <-30	Urea 22 mmol/L
Osmolality 412 mmol/kg	Creat 277 umol/L

What is the pH? 6.72 = Severe acidaemia

What is the primary process? HCO3 2 = Primary metabolic acidosis

Is there compensation?

Expected pCO2 **= 1.5 x HCO3 + 8**
= 1.5 x 2 + 8
= 11 mmHg
Measured pCO2 is 16 mmHg, supporting near maximal compensation

Are there other clues to diagnosis?

Corrected Na **= Na + (glucose – 5) / 3**
= 119 + (92 – 5) / 3
= 148 mmol/L

Anion Gap **= Corrected Na – (Cl + HCO3)**
= 148 – (98 + 2)
= 48
Consistent with a high anion gap acidosis

Delta Gap **= (Anion Gap – 12) ÷ (24 – HCO3)**
= (48 – 12) ÷ (24 – 2)
= 1.63
Consistent with a high anion gap acidosis without a co-existing non-anion gap component.

Calculated osmolality **= 2(Na) + urea + glucose**
= (2 x 148) + 22 + 92
= 410

Osmolar gap **= Measured osmolality – calculated osmolality**
= 412 – 410
= 2
Therefore normal osmolar gap.

Electrolyte Clues: Glucose is markedly elevated, serum sodium measures low, but is high when corrected for glucose. Serum potassium, urea and creatinine are all elevated. Chloride is within the normal range.

Expected PAO2 **= (713 x FiO2) – (pCO2 x 1.25)**
= (713 x 0.3) – (16 x 1.25)
= 193.9 mmHg

A-a gradient **= PAO2 – PaO2**
= 193.9 – 190
Therefore oxygenation is appropriate and there is no A-a gradient

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Formulation:

Description: This ABG shows a severe acidaemia with a high anion gap acidosis. There is no non-anion gap component. Near maximal respiratory compensation has occurred. Serum osmolality is markedly elevated, however there is no osmolar gap. Serum glucose is markedly elevated. Measured serum sodium is low, but when corrected for glucose the true sodium is high. Serum potassium, urea and creatinine are all elevated. Oxygenation is appropriate with no A-a gradient.

Interpretation: This ABG is consistent with a fully compensated severe diabetic ketoacidosis with HAGMA and elevated serum osmolality secondary to the high serum glucose (and to a smaller extent urea). Pseudohyponatraemia exists secondary to high glucose, and when corrected there is hypernatraemia, probably secondary to fluid loss / volume depletion. Potassium is elevated in keeping with acidosis. The renal failure may be chronic (diabetic nephropathy), and/or have a acute component secondary to dehydration.
Other causes of HAGMA (such as alcoholic ketoacidosis and toxic alcohol ingestion) would be less likely, although not completely excluded, in the presence of normal osmolar gap.

Additional information: Strong ion difference (Na-Cl=50) is consistent with a strong ion alkalosis, which further supports severe volume depletion.

ABG OF THE WEEK #07

Describe and interpret this venous blood gas:

History: 24y female who presents to ED feeling weak and unwell. Her clothes and hands are covered in paint. Her VBG on presentation is below.

pH 7.37	Na 137 mmol/L
pCO2 30 mmHg	K 2.5 mmol/L
HCO3 16 mmHg	Cl 121 mmol/L
Lactate 1.1	Glucose 10 mmol/L

ABG OF THE WEEK #07 - Answer

Describe and interpret this venous blood gas:

History: 24y female who presents to ED feeling weak and unwell. Her clothes and hands are covered in paint. Her VBG on presentation is below.

pH 7.37	Na 137 mmol/L
pCO2 30 mmHg	K 2.5 mmol/L
HCO3 16 mmHg	Cl 121 mmol/L
Lactate 1.1	Glucose 10 mmol/L

What is the pH? 7.37 = Normal pH (tending to acidaemia <7.4)

What is the primary process? HCO3 16 = Primary metabolic acidosis

Is there compensation?

Expected pCO2 **= 1.5 x HCO3 + 8**
= 1.5 x 16 + 8
= 32 (+/-2)
Actual pCO2 is 30, therefore fully compensated

Are there other clues to diagnosis?

Anion gap **= Na – (Cl + HCO3)**
= 137 – (121 + 16)
= 0
Consistent with non anion gap acidosis.

Electrolyte clues: Chloride is elevated, sodium is normal, potassium is low. Glucose is slightly elevated. Lactate is normal

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Formulation:

Description: This VBG shows a fully compensated primary non-anion gap acidosis with correction of pH to within normal limits. This is associated with moderate hypokalaemia, normal serum sodium and elevated chloride. Glucose is mildly elevated.

Interpretation: Elevated chloride is usually associated with a non-anion gap acidosis. Given the stated history, the most likely cause of this presentation is volatile solvent abuse which causes a type 1 distal renal tubular acidosis with potassium wasting and hyperchloraemic metabolic acidosis. Other electrolyte

abnormalities that should be considered include hypomagnesaemia, hypocalcaemia and hypophosphataemia. Differential diagnosis would include recent diarrhoea with GI bicarbonate and potassium loss, medications with carbonic anhydrase activity (acetozolamide, topiramate – often associated with hyponatraemia), pancreatic or small bowel fistulae. Adrenal insufficiency is less likely as this is normally associated with hyponatraemia and hyperkalaemia rather than hypokalaemia as seen in this patient.

Additional information: SID (Na – Cl = 137 – 121 = 16) supports a low SID non-anion gap metabolic acidosis. Toluene is metabolised to hippuric acid which may contribute to metabolic acidosis, crystalluria and acute tubular necrosis.

ABG OF THE WEEK #08

Describe and interpret this arterial blood gas:

History: 60y male presents with abdominal pain, and altered bowel habit.

pH 7.42	Na 133 mmol/L
pCO2 28.2 mmHg	K 4.1 mmol/L
pO2 221 mmHg	Cl 107 mmol/L
HCO3 18.1 mmol/L	Glucose 4.5 mmol/L
BE -4.9	Lactate 0.7 mmol/L

ABG OF THE WEEK #08 - Answer

Describe and interpret this arterial blood gas:

History: 60y male presents with abdominal pain, and altered bowel habit.

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pH 7.42	Na 133 mmol/L
pCO2 28.2 mmHg	K 4.1 mmol/L
pO2 221 mmHg	Cl 107 mmol/L
HCO3 18.1 mmol/L	Glucose 4.5 mmol/L
BE -4.9	Lactate 0.7 mmol/L

What is the pH? 7.42 = Normal range, tending to alkalaemia [>7.4]

What is the primary process? pCO2 28.2 = Primary respiratory alkalosis

Is there compensation?

Expected HCO3 **= 24 – 2 x {(40 – Measured pCO2)/10}**
= 24 – 2 x {(40 – 28.2)/10}
= 21.6 mmol/L
Measured bicarb is actually lower, therefore there is a coexistent primary metabolic acidosis.

Expected pCO2 **= 1.5 x HCO3 + 8**
= 1.5 x 18.1 + 8
= 35.15 mmHg
Measured pCO2 is lower, confirming coexisting primary respiratory alkalosis

Are there other clues to diagnosis?

Anion gap **= Na – (Cl + HCO3)**
= 133 – (107 + 18)
= 8
Suggests non-anion gap metabolic acidosis

Delta gap **= (AG – 12) ÷ (24 – HCO3)**
= (8 – 12) ÷ (24 – 18)
= -0.66
Consistent with non anion gap acidosis

Formulation:

Description: This arterial bloods gas shows a primary non-anion gap metabolic acidosis and coexisting primary respiratory alkalosis with overall pH within the normal range. Sodium is marginally below the normal range, but potassium, chloride and random glucose are all normal. Lactate is low. pO2 of 221 suggests the presence of supplemental oxygen delivery, but an A-a gradient cannot be calculated from the information available.

Interpretation: Non anion gap acidosis may be due to diarrhoea in context of altered bowel habit, differential diagnosis may include pancreatic fistula, adrenal insufficiency (given hyponatraemia) and carbonic anhydrase inhibitors. Respiratory alkalosis may be due to pain, anxiety or early sepsis (although low lactate is a good prognostic factor). These abnormalities would be expected to resolve with supportive treatment for underlying cause.

Additional information: Lipase 1100, CT abdomen showed “bulky pancreas” with changes consistent with acute pancreatitis.

ABG OF THE WEEK #09

Describe and interpret this arterial blood gas:

History: 58y female with a history of type 2 diabetes mellitus (treated with metformin) presents with reduced conscious state.

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pH 7.11	Na 136 mmol/L
pCO2 13 mmHg	K 5.1 mmol/L
pO2 120 mmHg (FiO2 0.21)	Cl 103 mmol/L
HCO3 4 mmol/L	Glucose 41 mmol/L
	Urea 16 mmol/L

ABG OF THE WEEK #09 - Answer

Describe and interpret this arterial blood gas:

History: 58y female with a history of type 2 diabetes mellitus (treated with metformin) presents with reduced conscious state.

---

pH 7.11	Na 136 mmol/L
pCO2 13 mmHg	K 5.1 mmol/L
pO2 120 mmHg (FiO2 0.21)	Cl 103 mmol/L
HCO3 4 mmol/L	Glucose 41 mmol/L
	Urea 16 mmol/L

What is the pH? 7.11 = Acidaemia

What is the primary process? HCO3 4 = Primary metabolic acidosis

Is there compensation?

Expected pCO2 **= 1.5 x HCO3 + 8 (+/-2)**
= 1.5 x 4 + 8
= 14 mmHg
Consistent with full respiratory compensation.

Are there other clues to diagnosis?

Corrected sodium **= Na + (glucose – 5)/3**
= 136 + (41 – 5)/3
= 148 mmol/L
Corrected sodium is elevated

Anion Gap **= Na – (Cl + HCO3)**
= 148 – (103 + 4)
= 41
Therefore a high anion gap metabolic acidosis

Delta gap ratio **= (AG – 12) ÷ (24 – HCO3)**
= (41 – 12) ÷ (24 – 4)
= 1.45
Suggests a high anion gap metabolic acidosis without an additional non anion gap acidosis

Potassium is at upper limit of normal
Serum glucose is markedly elevated

Expected PAO2 **= 150 – (pCO2 x 1.25)**
= 150 – (13 x 1.25)
= 133 mmHg

A-a gradient **= PAO2 – PaO2**
= 133 – 120
= 13

Maximum A-a gradient **= Age/4 + 4**
= 58/4 + 4
= 18.5
Therefore A-a gradient is not elevated

Formulation:

Description: This ABG shows a fully compensated, high anion gap metabolic acidaemia in the context of elevated blood glucose. There is no additional non-anion gap acidosis. Serum sodium measures within normal range, but is elevated when corrected for glucose. Serum potassium is marginally elevated, as is urea. Chloride is normal. There is no significant A-a gradient.

Interpretation: This gas suggests diabetic ketoacidosis, even though the patient is known to be a type 2 diabetic. There is probably volume contraction and dehydration with elevated urea and corrected sodium. Urine and/or serum ketones should be tested, and a search for underlying pathology (such as sepsis) may be required. A significant differential diagnosis would be a metformin-induced lactic acidosis which could present with a similar picture, and lactate should be measured. Volume replacement, insulin and potassium supplementation are required. Haemodialysis will be required if lactate is elevated, particularly if there is renal dysfunction.

Additional information: Lactate was 0.7, excluding metformin-induced lactic acidosis. C-peptide was 0.1, suggesting developing pancreatic failure and conversion to type 1 diabetes (reduced insulin production).

ABG OF THE WEEK #10

Describe and interpret this arterial blood gas:

History: 68 year old female presents confused and febrile. She appears tachypnoeic and is dehydrated. Her past medical history includes chronic back pain.

---

pH 7.48	Na^+ 142 mmol/L
pCO_2 23 mmHg	K^+ 3.3 mmol/L
pO_2 117 mmHg (FiO2 0.21)	Chloride 108 mmol/L
HCO3 15 mmol/L	Glucose 5.2 mmol/L
Lactate 1.1	Creatinine 244 micromol/L

ABG OF THE WEEK #10 - Answer

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What is the pH? 7.48 = Alkalaemia

What is the primary process? pCO2 23 = Primary respiratory alkalosis

Is there compensation?

Expected HCO3 **= 24 – 2 x {(40 – Measured pCO2)/10}**
= 24 – 2 x {(40 – 23)/10}
= 20.6
Measured bicarb is lower (15 mmol/L)
Therefore a primary metabolic acidosis co-exists

Expected pCO2 **= 1.5 x HCO3 + 8 (+/- 2)**
= 1.5 x 15 + 8
= 30.5
Measured pCO2 is lower (22.8)
Supports co-existing primary respiratory alkalosis

Are there other clues to diagnosis?

Anion Gap **= Na^+ – (Cl^- + $HCO3^-$)**
= 142 – (108 + 15)
= 18
Consistent with elevated anion gap acidosis

Delta gap **= (AG – 12) / (24 – HCO3)**
= (18 – 12) / (24 – 15)
= 0.66
Delta Gap < 0.8
Therefore a non anion gap acidosis coexists.

Expected PaO2 = PiO2 – (PACO2/0.8)
= 150 – (23/0.8)
= 121 mmHg

A-a gradient = PaO2 – PAO2
= 121 – 117
= 4
Therefore there is no A-a gradient

Formulation:

Description: ABG shows an alkalaemia from a respiratory alkalosis with an additional mixed anion gap and non-anion gap metabolic acidosis. The pCO_2 is lower than would be expected for respiratory compensation, supporting a primary respiratory alkalosis. Oxygenation is appropriate with a normal A-a gradient. Bicarb is lower than would be expected as compensation for an acute respiratory alkalosis, therefore a primary metabolic acidosis exists. There is a high anion gap acidosis and a raised delta gap suggesting a coexisting non-anion gap acidosis. Creatinine is elevated. Lactate is mildly raised, potassium is mildly low. Sodium, chloride and glucose are within normal limits.

Interpretation: This mixed primary respiratory alkalaemia and metabolic acidosis is characteristic for salicylate poisoning. In this case, chronic (therapeutic) salicylism is likely in view of the history of age, chronic pain, altered mental state, fever and dehydration. Renal failure is likely to have an acute component secondary to dehydration and altered mental state (reduced intake). Treatment includes urinary alkalinisation +/- escalation to dialysis if the level does not fall or renal failure worsens. Urinary alkalinisation will have to be performed cautiously in view of the hypokalaemia and K replacement will be required.
Differential would include sepsis with tachypnoea and metabolic acidosis.

Additional information: A salicylate level was added, and was 454 mg/L (reference range 140 – 350 mg/L).

ABG OF THE WEEK #11 - Answer

Describe and interpret this arterial blood gas:

History: An 85y female from a nursing home presents with altered conscious state for 24 hours.

---

pH 7.38	Na 175 mmol/L
pCO2 48 mmHg	K 3.6 mmol/L
pO2 87.3 mmHg	Cl 133 mmol/L
HCO3 28 mmHg	Glucose 10.8 mmol/L
BE 2.4	Lactate 3.1 mmol/L

ABG OF THE WEEK #12 - Answer

Describe and interpret this arterial blood gas:

History: 70y female presents with fever, purpuric rash and altered conscious state. She has received pre hospital IV fluid resuscitation.

---

pH 7.13	Na 140 mmol/L
pCO2 30 mmHg	K 2.8 mmol/L
pO2 187 mmHg	Cl 120 mmol/L
HCO3 10 mmHg	Glucose 2.6 mmol/L
Lactate 8.8 mmol/L	

ABG OF THE WEEK #2012.2.01

Describe and interpret this arterial blood gas:

History: A 26 year old male presents to ED with a 3 day history of vomiting. He had a similar presentation 6 months ago.

pH 7.68	Na 135 mmol/L
pCO2 55 mmHg	K 4.5 mmol/L
pO2 80 mmHg	Cl 48 mmol/L
HCO3 65 mmol/L	Glucose 4.5 mmol/L
BE 40	Urea 35 mmol/L
Lactate 14.9 mmol/L	Creatinine 656 mcmol/L

ABG OF THE WEEK #2012.2.02

Describe and interpret this arterial blood gas (FiO2 0.4):

History: An 18 year old male is brought to ED by ambulance following a severe motorbike accident. He has head, pelvis and leg injuries.

---

pH 7.01	Na 140 mmol/L
pCO2 77 mmHg	K 4.1 mmol/L
pO2 103 mmHg	Cl 114 mmol/L
HCO3 19 mmol/L	Glucose13.6 mmol/L
Lactate 5.9 mmol/L	

ABG OF THE WEEK #2012.2.03

Describe and interpret this venous blood gas:

History: 45 year old male presents following occupational exposure to inhaled hydrofluoric acid fumes (FiO_2 = 0.21). He complains of tingling in his fingers.

---

pH 7.46	Na 137 mmol/L
pCO_2 33 mmHg	K 4.3 mmol/L
pO_2 173 mmHg	Cl 109 mmol/L
HCO_3 23 mmol/L	Glucose 5.2 mmol/L
BE 0	Ionised Ca 1.12 mmol/L

Blood Gas Analysis

Acid-Base Calculations

Normal Ranges

pH = 7.35 – 7.45
PCO2 = 35 – 45 mmHg
HCO3 = 22 – 26
Anion gap = 6 – 12 mEq/L

1. Acidosis or alkalosis?

- Acidaemia is pH < 7.35
- Alkalaemia is pH > 7.45

pH does not completely normalise unless two pathologies are present.

2. Respiratory or metabolic?

Respiratory acidosis has PCO2 > 44 mmHg
Respiratory alkalosis has PCO2 < 40 mmHg

Metabolic acidosis has HCO3 < 25 mmHg
Metabolic alkalosis has HCO3 > 25 mmHg

3. Anion gap

AG = Na – (Cl + HCO3)

Normal AG
= 2-10 mEq/L (Tox handbook)
= 6-12 mEq/L (Dunn)

Correct for hypoalbuminaemia

- Add 2.5 mEq/L to the anion gap for every 10g/L albumin below 40 g/L.

4. Assess degree of compensation

Use these values

- pH = 7.40
- HCO3 = 24
- PCO2 = 40
- Anion gap = 12

Metabolic acidosis

Winter's formula:
Expected PCO2 = 1.5 x HCO3 + 8 (±2)
Lower limit of compensation is PCO2 ~ 10 mmHg

Metabolic Alkalosis

Expected PCO2 = 0.7 x HCO3 + 20 (±2)
Upper limit of compensation is PCO2 ~ 60 mmHg

Quick Method for Metabolic Acidosis / Alkalosis
PCO2 = last 2 digits of the pH between pH 7.1 – 7.6

Respiratory acidosis

Every 10 mmHg increase in PCO2 should increase the [HCO3-] by
- 1 mEq/L (acute)
- 4 mEq/L (chronic)

Respiratory alkalosis

Every 10 mm Hg decrease in PCO2 should decrease the [HCO3-] by
- 2 mEq/L (acute)
- 5 mEq/L (chronic)

5. Calculate Delta gap (if metabolic acidosis present)

Decrease in bicarbonate minus increase in anion gap:
- (24 – HCO3) - (AG – 12)
- Raised delta gap indicates additional NAGMA

Other calculations

6. Osmolar gap

- Calculated osmolality = 2 x [Na+] + urea + glucose + (ethanol x 218)
- Osmolar gap (OG) = measured osmolarity – calculated osmolality
- Normal OG is < 10

7. Aa-gradient

Calculated PAO2 = (713 x FiO2) – (PCO2 / 0.8)

Room air
Calculated PAO2 = 150 – (PCO2 / 0.8)

Aa gradient = calculated PAO2 – measured PaO2

Normal Aa gradient

- < age / 4
- < 10 in the young
- < 20 in the elderly
- Increased in the supine patient (due to V/Q mismatch)

Interpretation

Raised osmolar gap:

Due to:

- Alcohols
- Ketones
- Sugars
- Lactate
- Proteins
- Lipids
- Excessively high levels of ions (mg, phos, ca)

Exogenous agents

- Toxic alcohols
    - Methanol
    - Ethylene glycol
    - Propylene glycol (diluent in diazepam, phenytoin)
- Acetone
- Mannitol

Non-toxicological

- Ketoacidosis
    - Diabetic
    - Alcoholic
- Severe lactic acidosis
    - Shock
    - Trauma and burns
- Chronic renal failure
- High lipids / protein / ions (e.g. Mg2+)

In suspected toxic alcohol poisoning, multiply the osmolar gap by the following conversion factor to get the estimated toxic alcohol level in mg/dL.

- Ethylene glycol x 6.2
- Methanol x 3.2

High anion gap (>12) metabolic acidosis

Lactate

- Poor tissue perfusion
  - Shock
  - Sepsis
  - DKA
- Poisoning
  - Metformin
  - Salicylates
  - Iron
  - Valproate
  - CO / CN
  - Isoniazid

Ketoacids

- DKA
- Alcoholic ketoacidosis
- Starvation ketosis

Renal Failure

- Phosphate, sulphate, various organic acids

Toxic alcohol metabolites

- *Formate* with methanol
- *Oxalate* with ethylene glycol

Or CATMUDPILES

Normal anion gap (6-12) metabolic acidosis

= *hyperchloraemic* metabolic acidosis.

Mechanisms
- Bicarbonate loss
- Chloride gain

Iatrogenic
- Normal saline hydration

GI losses
- Diarrhoea
- Cholestyramine
- Small bowel or pancreatic fistula
- Ileal conduit

Renal losses
- Renal tubular acidosis
- Carbonic anhydrase inhibitors
    - Acetazolamide
    - Topiramate

Acidifying agents (uncommon)
- Ammonium chloride

Or remember USED CARP

The top causes are diarrhea, normal saline and renal tubular acidosis.

Low anion gap (< 6 mEq/L)

- Low albumin
- High levels of unmeasured cations
    - Ca2+
    - Mg2+
    - Li+
    - Positively charged proteins in IgG myeloma
- Nitrites
- Falsely elevated chloride
    - Bromide
    - Iodine
    - High triglycerides
    - Propylene glycol

Metabolic alkalosis

- Due to loss of hydrogen and chloride ions.
- Usually due to **vomiting** or diuretics.

Addition of base

- Antacids
- Milk-alkali syndrome
- $NaHCO_3$ (transient)
- Citrate (massive blood transfusion, dialysis)

Chloride depletion

- Vomiting
- NG suction
- Diuretics
- Chronic hypercapnia (renal retention of $HCO_3 > Cl$)

Potassium depletion (kidney retains $K^+ > H^+$)

- Hyperaldosteronism
- Cushing's syndrome
- Kaliuretic diuretics
- Bartter's / Gitelman's syndromes
- Excessive licorice intake (glycyrrhizic acid)
- Severe potassium depletion

Other disorders

- Laxative abuse
- Severe hypoalbuminaemia

Respiratory Acidosis

Acute

- Airway obstruction
- Aspiration
- Bronchospasm
- Drug-induced CNS depression
- Hypoventilation (e.g. neuromuscular)

Chronic

- Lung disease
- Neuromuscular disease
- Obesity

Respiratory alkalosis

CNS-mediated hyperventilation

- Raised ICP
- Cerebrovascular accidents
- Psychogenic

Hypoxia-mediated hyperventilation

- Altitude
- Anaemia
- V/Q mismatch

Pulmonary

- CCF
- Mechanical hyperventilation
- Pneumonia
- Pulmonary embolism

Sepsis

Toxin-induced hyperventilation

- Nicotine
- Salicylate
- Xanthines

Causes of a raised lactate

Type A lactic acidosis = imbalance between oxygen supply and demand

- Reduced oxygen delivery
  - Shock
  - Severe hypoxia
  - Severe anaemia
  - Carbon monoxide poisoning

- Excessive oxygen demand
  - Seizure
  - Hyperpyrexia
  - Exercise
  - Shivering

Type B lactic acidosis = metabolic derangement

Mnemonic = "BLACK MIST"

- Beta-2 agonists (salbutamol, adrenaline)
- Liver failure
- Alcohols (ethanol, methanol, ethylene glycol)
- Cyanide poisoning
- Ketoacidosis
- Metformin
- Inborn errors of metabolism
- Sepsis
- Thiamine deficiency