An approach to arterial blood gas analysis

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Introduction

A balanced acid-base status is critical for regular cellular and organ function and the survival of an organism. An arterial blood gas (ABG) machine is an accessible bedside tool of measurement, accessing both respiratory and metabolic disorders. Correct sampling, avoidance of common analytical errors, and correct interpretation of results are critical in guiding the administration of appropriate therapy. The adage "never do the test if you are not sure what to do with the results" is relevant concerning ABG analysis.

Historically and traditionally, the lungs and the kidneys have been considered important organs regulating acid-base balance. Next to these organs, the liver is now recognised as an important contributor to acid-base homeostasis. Understanding the collaboration, metabolic communication, and physiological interactions of these organs is crucial in understanding any acid-base derangement. These organs are the most important effectors, normalising the acid-base status.

An excess of metabolic acid is converted to volatile acid (CO_2) by the respiratory metabolic link and excreted by the lungs. Likewise, an excess of volatile acid is converted to metabolic acid and excreted by the kidney. This system is non-saturable. The hepatorenal link regulates the amount of bicarbonate generated or consumed, based on the alkalinity or acidity of arterial blood.

Physiological roles of organs in acid-base regulation

Lungs

Peripheral receptors, located in the aortic arch and carotid body, respond to acute changes in the arterial partial pressure of carbon dioxide ($PaCO_2$) and partial pressure of oxygen (PaO_2). In addition, carotid body chemoreceptors are sensitive to acute arterial blood pH changes.

Central chemoreceptors residing in the medulla are sensitive to acute changes in pH (H^+) in the extracellular fluid in the brain. These receptors also receive afferent input from the periphery via glossopharyngeal and vagus nerves. Central sensors linked to the respiratory centre, when activated, generate feedback
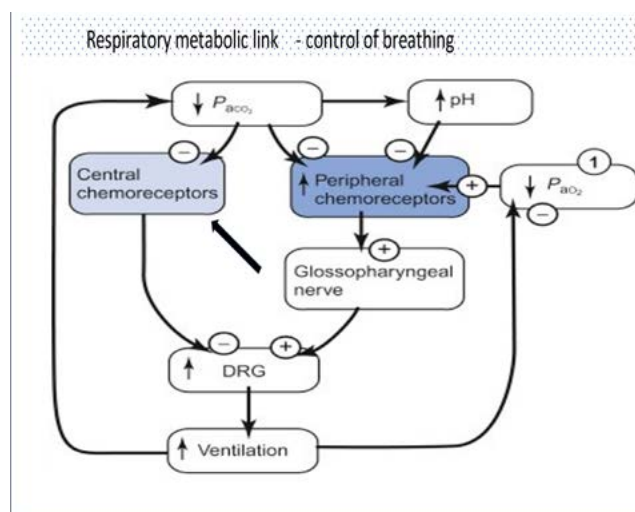

Figure 1: Chemical control of breathing and feedback loops. (DRG dorsal respiratory group)

mechanisms (negative or positive) to alter the respiratory pattern (Figure 1).

Cerebrospinal fluid (CSF) lacks the buffering capacity as little protein exists in the fluid. Therefore, a slight change in CSF PCO_2 results in a dramatic change in pH. Central correction is more rapid and effective than peripheral correction.

Liver

Lactate metabolism

Humans generate about 0.8 mmol/kg/hour of lactate and the healthy liver is the main consumer of lactate. About 30–70% of lactate is metabolised in the liver. It is converted to pyruvate and retransformed to glucose.

Albumin synthesis

In physiological pH, albumin behaves as a weak non-volatile acid. Its histidine residue donates a proton in a solution. Hypoalbuminemia represents a lack of acid and results in metabolic alkalosis hence base excess (BE) should always be corrected for albumin.
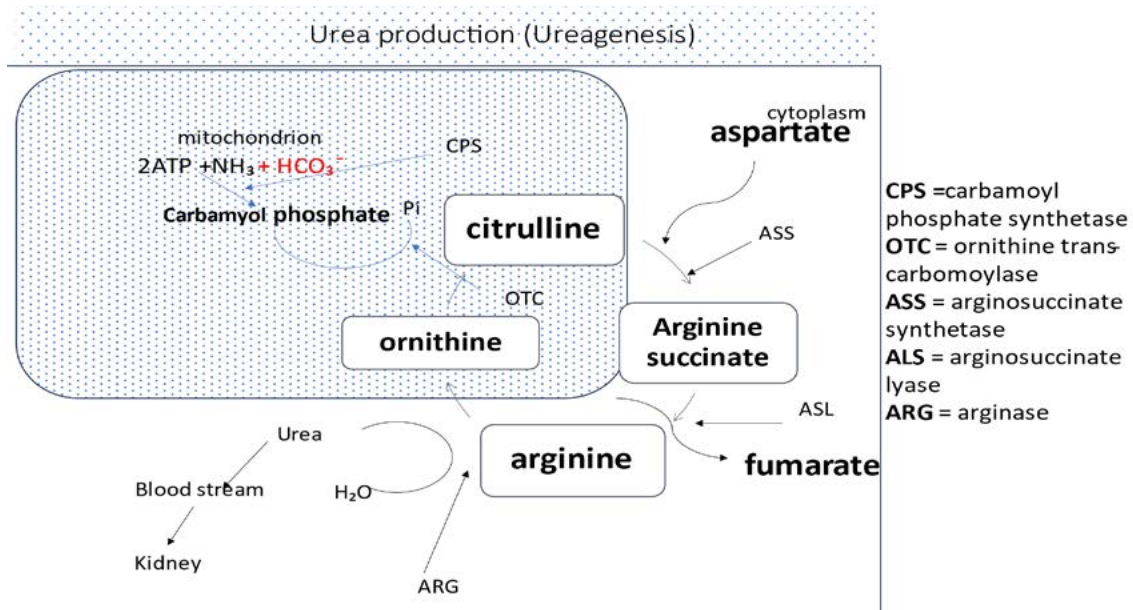
Figure 2: Hepatic urea synthetic pathway

- A 1 g/L decrease in albumin is associated with a 3.7 mEq/L increase in BE.

- A 1 g/L decrease in albumin is associated with a 0.25 mEq/L decrease in anion gap.

Hypoalbuminemia underestimates an anion gap.

Ketogenesis and ketoacids

Ketogenesis, in hepatic mitochondrial cells, is the salvage pathway for acetyl-coenzyme A, i.e. when carbohydrates and fat are incompletely oxidised. Ketoacids, 3-hydroxybutyrate, acetoacetic acid, and acetone dissociate in physiological pH to produce hydrogen ions. This may lead to ketoacidosis. Net production of ketones, as well as urinary excretion, is controlled by feedback mechanisms leading to reduced endogenous acid production if the pH decreases, and increased ketone production if the pH increases. This rapid up- and downregulation applies to both hepatic ketogenesis and lactate production. Hepatic ketogenesis is negligible and does not cause significant acidosis in normal conditions. However, starvation, massive alcohol consumption, or defective glucose transport can cause ketogenesis and substantial metabolic acidosis. Ketone bodies are utilised by extrahepatic cells, the brain, and the heart as an energy source.

Urea production

Neurotoxic weak acid $NH4^+$ arises during protein breakdown. NH_3 is processed in the liver (urea cycle). For every 1 mol of urea
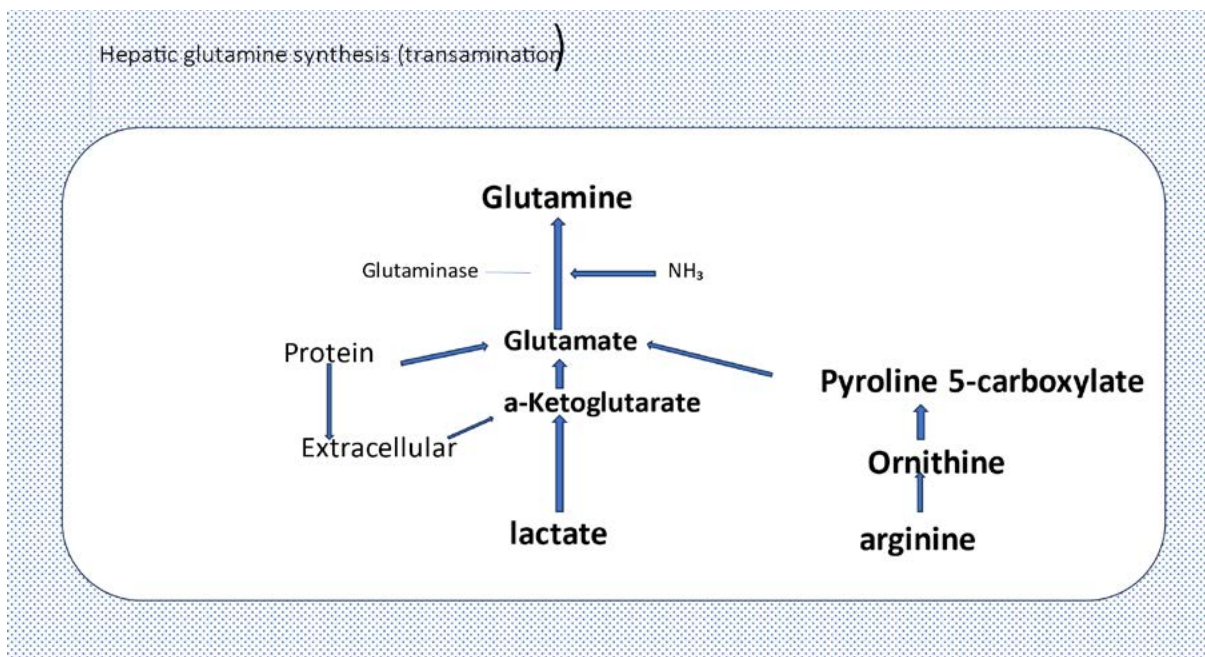

Figure 3: Hepatic transamination

generated, 1 mol of bicarbonate is consumed. This is an acidifying process (Figure 2 above). In acute metabolic acidosis, NH_3 is consumed (transamination) to generate glutamine instead of urea (Figure 3 above). Glutamine is subsequently taken up by the proximal convoluted tubules (PCTs) and catabolised to generate bicarbonate and glucose.

Transamination for glutamine synthesis (illustrated in Figure 3 below)

Alkalinising factors in patients with liver disease

In chronic liver disease, mixed respiratory and metabolic alkalosis are the main acid-base disorders.

- Ascites/hepatic hydrothorax → hypoxaemia and hyperventilation.
- Hyperammonemia and hepatic encephalopathy induce hyperventilation.
- Hepatopulmonary syndrome/portopulmonary hypertension aggravates dyspnoea.
- Secondary hypoaldosteronism induces metabolic alkalosis.
- Hypoalbuminemia is metabolic alkalosis.

Kidneys

The biochemistry of the ultrafiltrate in the PCTs is the same as in plasma. All components of the plasma are filtered through, except blood and high molecular weight substances. It is the function of the tubules to reclaim filtered substances. In Figure 4 below, sodium is reclaimed at the expense of urine acidification. The bicarbonate derived from peritubular carbonic anhydrase activity is reabsorbed with sodium through the $Na-HCO_3$ symporter system. There is no loss nor gain of any substance.

However, in acute metabolic acidosis, bicarbonate is preserved, and the conjugate base is filtered instead (B^-). There is a net gain of bicarbonate (generation) and a loss of a proton.

In addition, partial restoration of the acid-base balance is achieved through increased renal ammonia genesis and gluconeogenesis from plasma glutamate; and catabolism of glutamine (Figure 5 below).

Within 1–3 hours of acute acidosis, there is a 2–3 multiplied increase in hepatic glutamine synthesis (transamination). The following activities are accelerated:

- Glutamine is taken up by peritubular cells of PCTs from both circulation and luminal fluid. A specific transport system facilitates absorption from the venous site.
- Activation of apical Na/H antiport, which leads to the generation of bicarbonate. The Na/H antiport system also promotes the reabsorption of bicarbonate on the basolateral site.
- Glutamine is taken up by mitochondria and subjected to mitochondrial enzymatic activities. Glutaminase deaminates glutamine to glutamate, and glutamate is further deaminated by dehydrogenase to form α-ketoglutarate. In both processes, a molecule of ammonia is generated.
- α-Ketoglutarate enters the tricyclic acid (TCA) cycle with a subsequent generation of 2 mol bicarbonate.

Analysing the above processes:

- 1 mol HCO_3^- is generated through acidification of urine (Na/H antiport activity).
- 2 mol HCO_3^- is generated per mol of α-ketoglutarate.
- 2 mol NH_3 is generated by the mitochondrial deamination process.
- Hydrogen is excreted as ammonia salts ($-NH4^+$).
- Activation of the $Na-HCO_3^-$ symporter system promotes the reabsorption of the product.
- In the distal convoluted tubules, HCO_3^- is preferentially reabsorbed in exchange for other anions.

In chronic acidosis, some of the acute adaptations are partially compensated and the plasma glutamine falls to about 70%. The kidney, however, continues to extract more than a third of glutamine. Renal glutamine catabolism is sustained due to increased expression of genes encoding mitochondrial
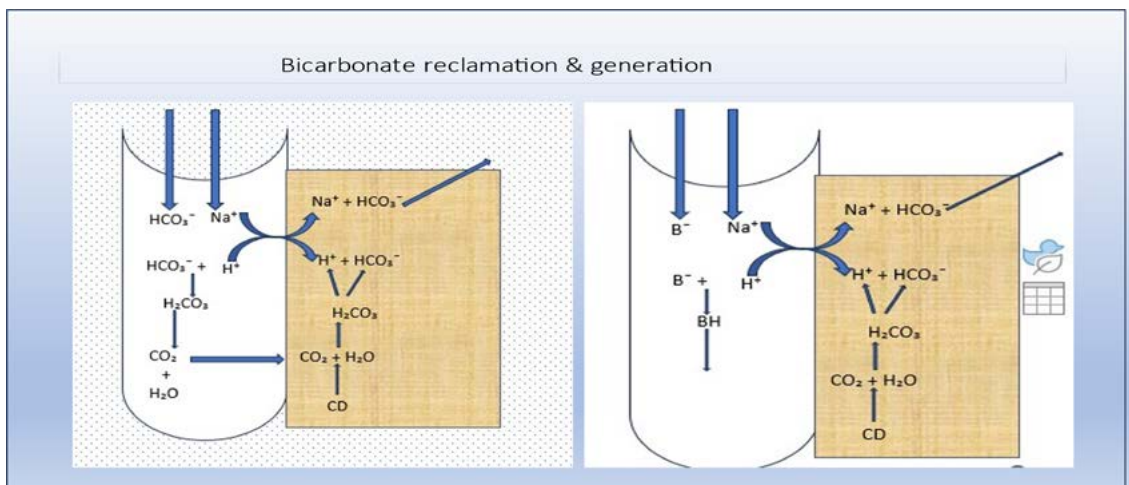

Figure 4: Bicarbonate reclamation and generation
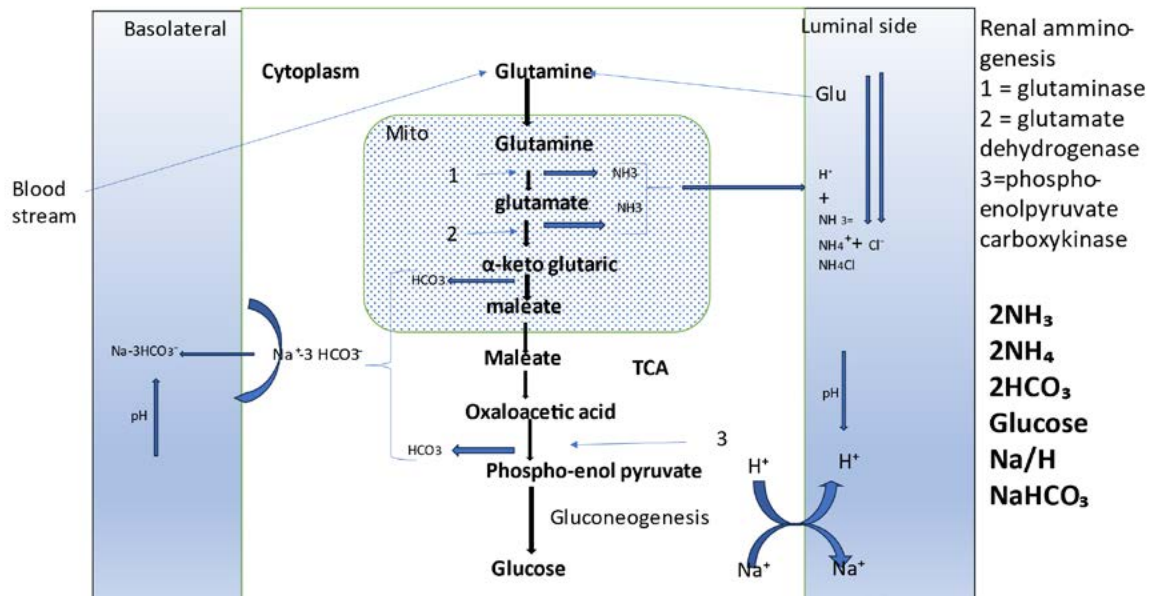
RENAL AMMONIAGENESIS

Figure 5: Ammonia genesis

glutaminase and glutamine dehydrogenase, cytoplasmic phosphoenolpyruvate carboxykinase (PEPC), apical Na/H antiport, and basolateral Na-bicarb symporter systems.

In addition, phosphate becomes important as a buffering agent.

Definition of concepts and useful terminology

Acid

A substance that donates a proton in a solution. A strong acid completely transfers its protons to water, leaving no undissociated molecules in a solution. Its conjugate base has a negligible tendency to be protonated (to abstract protons) in an aqueous solution. Weaker acids only partially dissociate in aqueous solutions and, therefore, exist in the solution as a mixture of acid molecules and their constituent ions. Strong acids have their equilibria shifted towards the right and thus have larger acidity constants (K_a); weaker acids have their equilibria shifted towards the left and smaller acidity constants. The strength of an acid depends on the number of protons it can donate.

Base

An acceptor of a proton in a solution.

A protein molecule can be both an acid and a base because proteins can accept an acid and donate a base.

Alkali

A substance that can donate a hydroxyl ion and can also accept a proton in a solution. All alkalis are bases, but not all bases are alkalis.

Standard bicarbonate

The concentration of bicarbonate in a sample of plasma should be kept under precise "standard" conditions, i.e. 37 °C and $PaCO_2$ of 40mmHg or 5.3 kPa. These changes are chosen to eliminate the respiratory contribution to acid-base status.

Actual bicarbonate

A concentration of bicarbonate measured in plasma without correction. It reflects the contribution of both respiratory and metabolic components of acid-base balance.

Base excess/deficit

The amount of strong univalent acid (HCL) or base (NaOH) required to titrate 1 L of blood back to pH 7.40. BE and deficit considers all buffers in the blood sample and is therefore considered a more accurate assessment of the metabolic component of the patient's acid-base status.

Buffers

Special protein or enzyme molecules function like a sponge to "mop up" hydrogen ions. They work effectively at very narrow margins. Humans have buffers operating in both intracellular and extracellular environments. Extracellular buffer proteins include the carbonic/bicarbonate system, phosphates, and ammonia.

Acidaemia and alkalaemia

These concepts indicate disturbances of pH in extracellular fluid. Alkalaemia: pH > 7.40. Acidaemia: pH < 7.40.

Acidosis and alkalosis

Refer to processes occurring at the cellular level that, if left uncorrected, would result in pH changes (acidaemia or alkalaemia). Acidosis/acidaemia and alkalosis/alkalaemia cannot be used interchangeably. Acidosis and alkalosis may still apply even if the pH is normal. For example, the patient may be simultaneously producing excessive but equal amounts of an acid and base, such that they balance each other out. In this case, the pH will be normal, but the underlying pathophysiological processes will still be present.

"Acidosis can exist without acidaemia, but acidaemia cannot prevail without acidosis."

Avoid sampling and analysing pitfalls.

- Obtain blood samples aerobically and aseptically.

- In the presence of an indwelling arterial catheter, remove about 5 ml of flushing solution from the cannula before actual sampling.

- Heparinise the sampling syringe to protect the microtubes of the ABG machine from clotting.

- Heparin is an acid and too much of it would influence the determination of $[H^+]$ and therefore pH levels.

- The presence of air bubbles allows CO_2 and O_2 to diffuse in or out of the bubbles and alter results within minutes, especially during transportation.

- The sample must be analysed at the patient's temperature – the results are always corrected for temperature.

  ◦ Henry's law. The amount of gas going into a solution is inversely proportional to temperature. In hypothermia, more gases would go into a solution, especially carbon dioxide, thus giving an alkalotic picture. However, total carbon dioxide will be the same.

  ◦ Temperature and vapour pressure. A direct relationship exists between the two. The higher the temperature, the higher the vaporisation process and the lower the partial pressure of gases.

  ◦ pKa and temperature chemical reaction. Temperature affects the rate of chemical reactions of the buffering solutions.

- The sample should be promptly analysed. If there is a delay in sample analysis, keep the sample on ice to slow down the rate of metabolism (can be delayed up to one hour).

- Put a stopper or cap to prevent arterial blood from interacting with oxygen in the air.

Blood gas machine

- A box full of electrodes arranged in a specific order. Each electrode is designed to perform specific functions and they are governed by different physical principles and laws of physics (beyond the scope of this talk).

- The average shelf life of electrodes is 6–12 months, depending on how often they are used.

- Electrodes should be regularly inspected for protein and debris accumulation as well as the integrity of protective plastic membranes.

- Daily calibration must be done for correct results.

- The incorporated automatic temperature calibration system must be kept intact.

Arterial blood gas analysis and interpretation

Be systematic and group parameters belonging together.

Oxygenation adequacy (indices of oxygenation)

Tension-based indices

Arterial partial pressure of oxygen (PaO_2).

PaO_2 alone has no value. Relate it to inspired oxygen concentration (FiO_2). A patient registering a PaO_2 of 100 mmHg at 100% oxygen is relatively hypoxemic. PaO_2 can also be used as a function of oxygen cascade: delivery of oxygen to the lungs (respiratory mechanics), transfer of oxygen to the blood (diffusion laws), carriage of oxygen by the blood (oxygen-haemoglobin dissociation curve), delivery of oxygen to the tissues (DO_2), release of oxygen to the tissues (oxygen-haemoglobin dissociation curve and Bohr effect), and return of oxygen to the lungs (mixed venous sats).

PA-aO2 (Alveolar-arterial oxygen tension difference):

- Normal PA-aO2 (extra-alveolar causes): hypoventilation, low FiO_2, low barometric pressure (high altitude), ventilator-related.

- High PA-aO2 (intra-alveolar causes): intrapulmonary shunt, diffusion defect, dead space ventilation, increased peripheral oxygen extraction.

PaO_2/FiO_2 ratio. Acute lung injury (ALI) and acute respiratory distress syndrome (ARDS). Familiarise yourself with the diagnostic criteria of both.

Oxygen index (OI)
OI= FiO_2 × mean arterial pressure (MAP) / PaO_2. Takes into consideration respiratory mechanics. Mean airway pressure: 25% good, 25–40% high mortality, and = 40% needs extracorporeal membrane oxygenator (ECMO).

Respiratory index (RI)
RI= PA - aO2 / PaO_2. Normal is > 1, 1–5 is V/Q mismatch, < 5 is a physiological shunt.

Content-based indices

Venous admixture (Qs/Qt):

$$Qs = \frac{Cc'O_2}{Cc'O_2} - \frac{CaO_2}{CvO_2}$$

Co-oximetry:

- HBO2. Haemoglobin dissociation curve, haemoglobin concentration, type of haemoglobin, and environment in which the haemoglobin operates.

- COHB. Cigarette smoking and environmental pollution/ automobile exhaust emissions. A small percentage is a product of extravascular haemolysis.

- MetHB. Oxidized haemoglobin. It is normally kept under 1%.

- Fetal haemoglobin (FFHB).

Carbon dioxide

Adequacy of respiration. The balance between the production and elimination rates.

Electrolyte analysis

This is a topic on its own and beyond the scope of this talk. However, it is worth mentioning here, based on clinical practice that metabolic alkalosis causes hypokalaemia and severe hypokalaemia sustains metabolic alkalosis. It may be extremely difficult to correct hypokalaemia despite copious administration of potassium, without addressing causes of metabolic alkalosis.

Metabolic alkalosis causes hypokalaemia:

- At cellular level: Electrolyte shift. Intracellular hydrogen is exchanged for extracellular potassium.

- Renal level: Na/K antiport promoted at expense of Na/H antiport system.

- Secondary hypoaldosteronism, due to contracted extracellular volume, promotes Na reabsorption at the expense of potassium.

Chronic hypokalaemia sustains metabolic alkalosis:

- Cellular level: Electrolyte shift. Intracellular potassium will be shifted in exchange for hydrogen. However, hydrogen is generated with bicarbonate.

- Chronic hypokalaemia stimulates PCTs to generate ammonia via glutamine activity. This generates bicarbonate.

Therapeutic classification of metabolic alkalosis:

- Saline responsive: U [Cl^-] < 10 mmol/L

  Vomiting, contracted extracellular fluid, preservation of chloride by the kidney. This alkalosis responds well to saline administration, unless on diuretics.

- Saline resistant: U [Cl^-] > 20 mmol/L

Familiarise yourself with the broad causes of metabolic alkalosis and acidosis and their physiological effects.

Indices of perfusion

BE, lactate, and mixed venous sats.

Analysis of metabolites

Lactate, glucose, and bilirubin (a topic on its own).

Acid-base status (HCO_3^- based vs. biochemical approach)

Henderson-Hasselbalch equation

This equation describes the relationship between pH, pK_a, and [acid] and [base] concentration:

$$pH = pK_a + \log \frac{[HCO_3^-]}{[H_2HCO_3]}$$

$$pH = pK_a + \log \frac{[HCO_3^-]}{[0.03 \times PCO_2]}$$

$$pH = 6.1 + \log \frac{[20]}{[0.03 \times 40]}$$

$$pH = 6.1 + 1.3 = 7.40$$

Take note:

- Ka is the dissociation constant of H_2HCO_3; pK_a is the negative common logarithm of Ka.

- The pH is the negative common logarithm of hydrogen ion concentration. Briefly, pH is proportional to bicarbonate and inversely proportional to PCO_2. However, bicarbonate is a dependent variable and its relationship with pH is non-linear.

- The emphasis is on the ratio of HCO_3^- / $0.03 \times PCO_2$ and not the individual components.

- If the ratio remains 20, the pH will be normal.

- The ratio forms the basis of compensation. An increase in one component should be associated with an increase in a second component by the same margin to keep the pH constant.

- Compensation dictates that changes should be in the same direction. A convergent or divergent picture suggests mixed acid-base status. Metabolic acidosis will be compensated by respiratory alkalosis. The opposite is also true.

- Compensation is never complete.

Stewart's approach to acid-base

An alternative approach to deciphering the complex acid-base status of patients. It takes into consideration the pathophysiology of diseases and organ interactions.

Principles:

- State of electroneutrality. Negatively charged ions are exactly equal to positively charged particles. An increase in one negatively charged particle, for example, will be accompanied by a decrease in another negative particle by the same margin or charge.

- Law of mass conservation: An amount of a substance in the body remains constant unless generated or removed.

  ◦ Routinely measured cations: Na/K/Ca/Mg.

  ◦ Unmeasured cations: proteins, lithium, and aluminium.

  ◦ Commonly measured anions: chloride and bicarbonate.

  ◦ Other anions: $SO4^{2-}$, H_2PO4^{2-}, and albumin.

  ◦ Organic anions: citrate, lactate, alcohol, and other toxins.

Fundamental differences between Stewart's approach and bicarbonate-centred models

- Hydrogen and bicarbonate are not independent determinants of acid-base status, but the result of changes in other systems. Hydrogen and bicarbonate ion concentrations are dependent on combined strong ion difference (SID), total weak acid concentration, and partial pressure of CO_2.

- BE is the overall measure of metabolic acid-base status. The amount of strong univalent acid (HCL) or base (NaOH) required to titrate 1 L of blood back to pH 7.40. Normal range: -3–3.

- Weak acids (albumin and phosphate) are also important in the determination of acid-base status. Phosphate participates more in chronicity. Weak acids play a role opposite to SID. Hyperalbuminemia ($\uparrow$ weak acid concentration) causes acidosis, while low albumin ($\downarrow$ total weak acids) induces alkalosis.

- The principal metabolic factor is the SID. Three independent controllers of acid-base status: CO_2, SID, and total concentration of weak acids.

  ◦ Strong ions dissociation completely in a solution, NA^+, K^+, Cl^-.

  ◦ Weak ions dissociation incompletely in a solution, HCO_3^-,

  ◦ $PO4^-$ and albumin.

  ◦ Lactate is regarded as a strong ion as it dissociates almost completely in a solution.

  ◦ Measured SID: Sum of all plasma cations minus anions that are measured routinely in practice.

  ◦ Na^+ is a strong cation while Cl^- is a strong anion in plasma, therefore SID = Na - Cl (40 - 42 mmol/L).

  ◦ $\uparrow$ SID suggest alkalosis, $\uparrow$ Na^+ or $\downarrow$ Cl^-, and therefore $\uparrow$ HCO_3^-.

  ◦ $\downarrow$ SID suggest acidosis, $\downarrow$ Na^+ or $\uparrow$ Cl^-, and therefore $\downarrow HCO_3^-$.

  ◦ In metabolic acidosis strong ions squeeze HCO_3 out, the opposite is true for alkalosis. For every 1 mEq/L in NACL difference, BE will change by 1 mEq/L in a negative direction for $\downarrow$ SID and positive for $\uparrow$ SID, i.e. in the same direction.

Calculated SID = $[Na^+ + K^+]$ - [albumin × 0.2 + phosphate × 1.5 + Cl^- + HCO_3^-]

Therefore, an overall BE:

- Lactate base effect = 2 - measured lactate.

- Albumin base effect = 0.25 (normal - measured albumin level).

- SID base effect = (Na^+ - Cl^- - 40).

Consider other changes in strong ions and weak acids, especially in organ dysfunction of the kidneys and liver. If the above three factors do not explain observed changes in the BE, then one or more other factors are involved.

Putting it all together

BE = [Na - Cl - 40] + [2 - measured lactate] + [0.25 (normal - measured albumin)] + OI

OI = BE - [Na - Cl - 40] + [1 - measured lactate] + [0.25 (normal - measured albumin)]

Practical exercise

An intubated and ventilated patient is transferred from the accident and emergency department for urgent laparotomy. Large bowel obstruction is the working diagnosis. The patient has had saline resuscitation.

Analysis of blood gas results:

Biochemistry: Na 145 mmol/L, K 3.0 mmol/L, Ch 117 mmol/L, albumin 28 g/L, lactate 7.0 mmol/L

ABG: pH 7.20, PCO_2 40 mmHg, bicarbonate 15 mmol/L, BE -14.5 mmol/L

Bicarbonate based approach.

- Acidaemia, significant base deficit. This suggests metabolic acidosis.

- Anion gap: (Na + K) - (HCO_3 + Cl^-) = (145 + 3) - (117 + 15) = 16. It appears as normal anion gap metabolic acidosis.

- However, bicarbonate and PCO_2 converge. This suggests mixed respiratory and metabolic acidosis. It cannot be a compensated picture.

- Which one is primary and secondary? Test it using Winter's formula.

  ◦ Expected PCO_2 = 1.5 (bicarbonate) + 8 = 1.5(15) + 8 = 30.5 mmHg. The measured PCO_2 is 40 mmHg. Therefore, it would appear that PCO_2 is elevated.

  ◦ Expected bicarbonate = 0.44 (PCO_2) + 7.6 = 24.5. The calculated bicarbonate is 15 mmol/L. A mixed picture would therefore make sense.

But chloride is high (117 mmol/L).

Stewart's approach (physical chemical approach)

Biochemistry: Na 145 mmol/L, K 3.0 mmol/L, Ch 117 mmol/L, albumin 28g/L, lactate 7.0 mmol/L

ABG: pH 7.20, PCO_2 40 mmHg, bicarbonate 15 mmol/L, BE -14.5 mmol/L

- BE = SID effect + lactate effect + weak acids effect + OI

- (Na – Cl - 40) + (2 - 7.0) + 0.25(40 - 28) + OI

- -12 + -2 = -14.0

- OI = -14.5 + 14 = -0.5 (insignificant contributor)

- Therefore:

  ◦ Change in BE is secondary to pure hyperchloremia.

  ◦ Partly offset by low albumen level.

  ◦ Partly aggravated by lactate.

  ◦ Insignificant contribution of OI to acid-base derangement.

See Table I - commonly used fluids in theatre.

Table I: Comparison of biochemistry of commonly used fluids and extracellular fluid compartment

Common fluids				
	ECF Compartment	Balsol	Ringers	Mod Ringers
Na⁺	142	130	131	131
K⁺	5	4	5.4	5.4
Mg⁺⁺	1	1.5		
Ca⁺⁺	2.5		1.8	
Cl⁻	105	110	112	108
HCO₃⁻	27	27		
Lactate			29	29
Osmolality	310	273	278	273

(Table I header and values, with superscripts:) Na$^+$, K$^+$, Mg^{++}, Ca^{++}, Cl$^-$, HCO$_3^-$

Treatment options of hyperchloremic metabolic acidosis using fluids in table I

PlasmaLyte (Balsol)

Na 145 mmol/L, K 3.0 mmol/L, Ch 117 mmol/L, albumin 28 g/L, lactate 7.0 mmol/L, SID = 28, BE = -14.5

PlasmaLyte:

- Na = 130 mmol/L and chloride 110 mmol/L
- Combined SID: (145 + 130) / 2 - (117 + 110) / 2
- 137.5 - 113.5 = 24
- 24 - 28 = -4
- Therefore, final BE: -4 + -14.5 = -18.5 (For every 1 mEq/L in NACL difference, BE will change by 1 mEq/L in the same direction.)

Ringer's lactate

Na 145 mmol/L, K 3.0 mmol/L, Ch 117 mmol/L, albumin 28 g/L, lactate 7.0 mmol/L, SID = 28, BE = -14.5

Ringer's lactate:

- Na 131 mmol/L and chloride 112 mmol/L
- Combined SID: (145 + 131) /2 - (117 + 112) / 2
- 138 - 114.5 = 23.5
- 23.5 - 28 = -4.5
- Final BE: -4.5 + -14.5 = -19.0

Half normal saline

Na 145 mmol/L, K 3.0 mmol/L, Ch 117 mmol/L, albumin 28 g/l, lactate 7.0 mmol/L, SID = 28, BE = -14.5

Half normal saline:

- Na 77 mmol/L and chloride 77 mmol/L
- Combined SID: (145 + 77) / 2 - (117 + 77) / 2
- 111 - 97 = 14
- Final BE: 14 - 28 = 14 = -14.5 + -14 = -28.5

Sodium bicarbonate (50 mEq/L)

Na 145 mmol/L, K 3.0 mmol/L, Ch 117 mmol/L, albumin 28 g/l, lactate 7.0 mmol/L, SID = 28, BE = -14.5

50 mEq/L sodium bicarbonate:

- Combined SID: (145 + 50) / 2 - (117 + 0) / 2 = 97.5 - 58.5 = 39 - 28 = 11
- Final BE: -14.5 + 11 = -3.5 (better)

Sodium bicarbonate (10 mEq/L)

Na 145 mmol/L, K 3.0 mmol/L, Ch 117 mmol/L, albumin 28 g/l, lactate 7.0 mmol/L, SID = 28, BE = -14.5

10 mEq/L sodium bicarbonate:

- Combined SID: (145 + 10) / 2 - (117 + 0) / 2 = 77.5 - 58.5 = 19
- 19 - 28 = -9
- Final BE: -14.5 + -9 = -23.5
- Hypo-osmolar bicarbonate

Pharmacological intervention?

- Forced diuresis in volume replete patient.
- Loop diuretics.

Reconstitute your own fluid.

You may also reconstitute your fluid, e.g. adding sodium to 5% dextrose water solution.

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