Postal Registration No. TN/CH/(C)/59/2023-2025
Registered News Paper Posted at Egmore R.MS. Patirika Channel.
Licensed to post without prepayment TN/PMG/(CCR)/WPP-500/2023-2025

RNI NUMBER : TNENG/2004/14197
RDISNo. 6342/98

Medi Quest BRS Hospital

A monthly News letter from BRS Hospital

UNTANGLING THE PUZZLE THAT IS METABOLIC ACIDOSIS.

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Price Rs. 5/- Only

November - 2023

Medi - 18

Quest - 2

Yearly Subscription

Rs 50/- only

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Metabolic acidosis is one of the most commonly encountered metabolic states in critical care practice. As a clinician it is important to recognise the signs of metabolic acidosis, be able to differentiate the various subtypes and also treat the underlying pathologies as needed.

What can cause metabolic acidosis?

Before we can talk about the subtypes of metabolic acidosis, it is important to understand what acidemia is and what acidosis is and how it occurs in our body. Acidemia is the state in which there is accumulation of excess acid in our body which leads to the fall in pH. Now acidemia can be due to metabolic causes or respiratory causes.

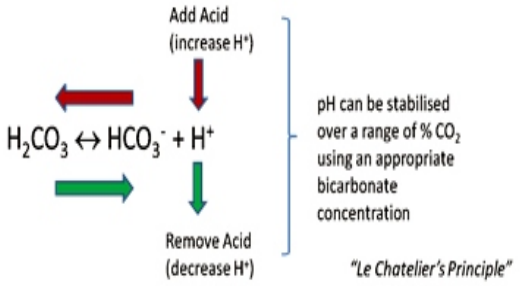
In order for metabolic acidosis to occur there has to be either increased levels of acid or reduced levels of base. This can happen in one of four mechanisms:

1. Increased acid production – eg DKA, Sepsis
2. Increased acid consumption – eg paracetamol, paraldehyde poisoning Salicylate, Methanol, ethylene glycol
3. Reduced acid excretion – eg renal tubular acidosis
4. Increased bicarbonate loss – eg diarrhea, renal tubular acidosis

The bicarbonate buffer system

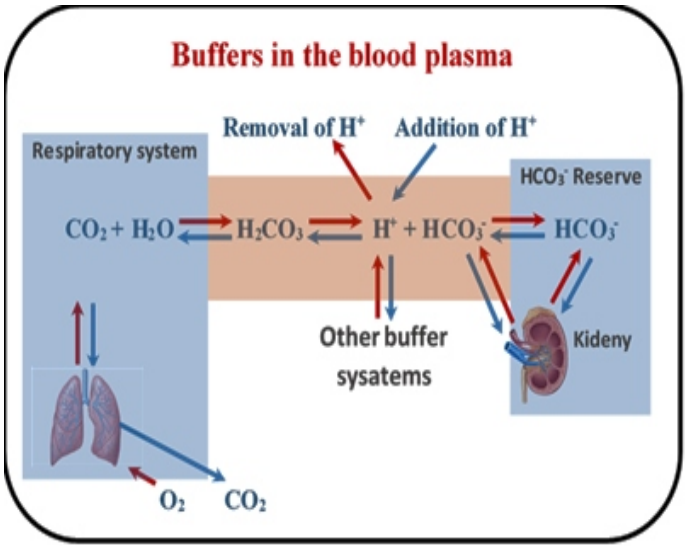
When there is acidemia, the acids are neutralised by the bases present in the blood. These neutralisation systems are known as buffer systems. The most important buffers in the blood are plasma proteins, phosphates and bicarbonate buffers. Of these, the most powerful buffer system is the bicarbonate / carbonic acid buffer system.

The currency for acid in our body is carbonic acid (H2CO3). Depending on what is needed, this carbonic acid is converted to bicarbonate and a proton or its converted into water and carbon dioxide and these reactions are fully reversible.



The above figure shows us what happens when there is increased acid in the blood. Acids are essentially proton [H+] donors. When they enter into the system, the dissociate into the negatively charged acid and the positively charged proton [H+]. The proton [H+] combines with the bicarbonate [HCO3-] to form

carbonic acid. This leads to a fall in the bicarbonate levels [HCO3-] which explains why in metabolic acidosis, bicarbonate [HCO3-] levels are always low. Carbonic acid is essentially inert but too much of it can cause problems and so the body needs a way of getting rid of it. This is where the respiratory compensation comes into play.



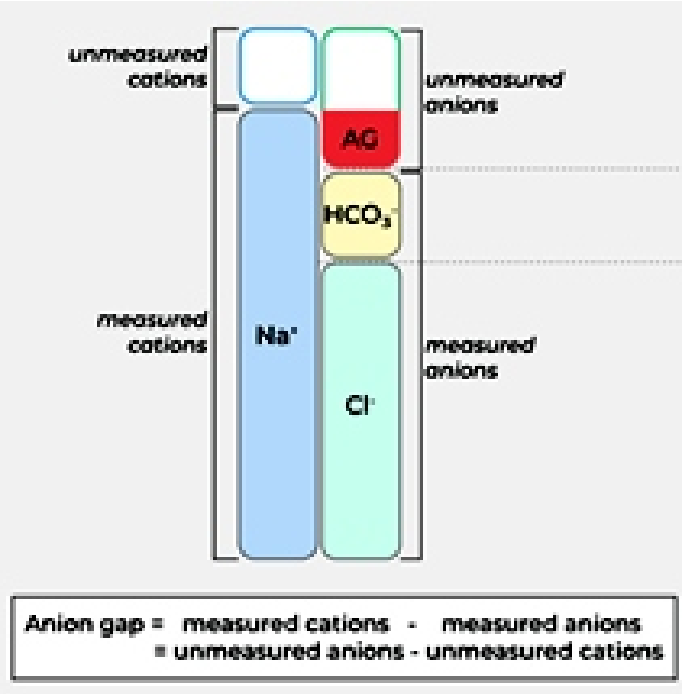
When the carbonic acid accumulates in the plasma, it is acted on by carbonic anhydrase and converted into carbon dioxide (CO2) and water (H2O). This CO2 is then breathed out and thus excess acid is eliminated from the system. In severe acidosis, the respiratory rate becomes rapid and shallow to allow for quick excretion of CO2. This pattern of breathing is known as Kussmaul's breathing.

Anion Gap

Now that we have an understanding of the buffer systems in the body, its important to understand when to use the buffer system to our advantage and when to add more. In the beginning we listed 4 mechanisms which can lead to acidosis. Out of those 4, the first three have to do with increasing acid levels in the blood. The last one has to do with a fall in the level of base. Physiologically, we are capable of handing increased acid in our system because we have this robust buffer system in place. However, loss of bicarbonate is harder for our body to handle.

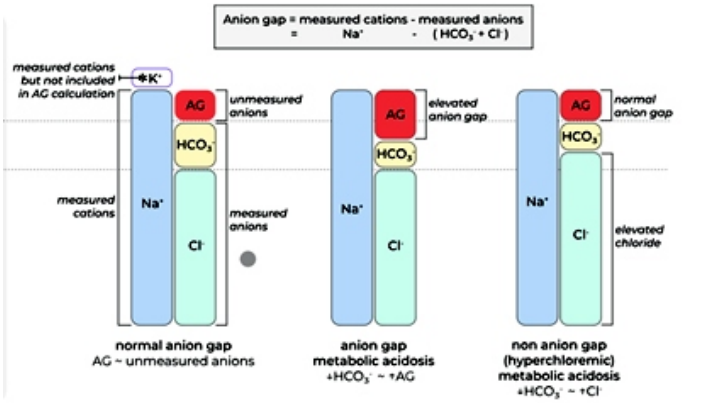
So the question is, how do we identify if the acidosis is due to excess acid or due to inadequate base?

This is where the anion gap comes in. Of the cations (positively charged ions), the ones we measure are sodium and potassium. Of the anions (negatively charged ions) the ones we measure are bicarbonate and chloride. But, in our system, there are small quantities of unmeasured cations and anions. The amount unmeasured anions is known as the anion gap.



Remember that acids dissociate into a negatively charged particle and a proton [H+]. So acids are all anions. This means that when there is an increase in the amount of acid, there is a rise in the amount of unmeasured anions. This leads to a higher anion gap which we refer to as high anion gap metabolic acidosis (HAGMA)

Conversely when there is a loss of base, there is reduced bicarbonate levels. These is compensated by increase chloride levels. This ensures that the anion gap remains untouched. This is referred to as normal anion gap metabolic acidosis (NAGMA)



HAGMA vs NAGMA and its Causes

Conceptually, the difference between the two is simple. High anion gap metabolic acidosis occurs because there is excessive acid in the system. Normal anion gap metabolic acidosis occurs because of loss of bicarbonate.

The causes of high anion gap metabolic acidosis (High levels of acid) are given by the mnemonic MUDPILES:

- M - methanol
- U – uremia
- D – Diabetic ketoacidosis / starvation ketoacidosis
- P – Paracetamol / Phenformin / palaldehyde
- I – Iron / Isoniazid
- L – Lactic acidosis
- E – Ethanol / ethylene glycol
- S – Salicylates / Asprin

The causes of normal anion gap metabolic acidosis (Low levels of bicarbonate) are given by the mnemonic USED PART

- U – Urinary diversion surgery
- S – small bowel fistula
- E – Extra chloride
- D – Diarrhea
- P – pancreatic fistula
- A – Addison's disease / Acetazolamide
- R – Renal tubular acidosis
- T – Tenofovir / Topiramate

Once we have identified the type of metabolic acidosis that is present, we will know if the situation is because of excess acid or inadequate bicarbonate. It is important to reiterate that in both cases, the serum bicarbonate levels will be low. In HAGMA, bicarbonate is low because its used up in the buffering of the excess protons [H+] released by the large amounts of acid. In NAGMA, bicarbonate is low because it is lost in urine or stools or both.

To give or not to give bicarbonate, that is the question

It would seem logical to give bicarbonate since all metabolic acidosis leads to a fall in the levels of bicarbonate. However bicarbonate on its own, can lead to a lot of issues. Complications that can be caused by bicarbonate are as follows:

1. Hypokalemia
2. Hypocalcemia
3. Prolonged QT interval
4. Hypercapnea
5. Hemodynamic instability
6. Increased urinary sodium excretion
7. Can cause vascular calcifications

So when do you give bicarbonate? The inherent buffer system that is present in the body is equipped to handle a fair amount of acidosis. Which means that the only time you need to give bicarbonate from an external source is if either the acidosis is so severe that our buffer systems are overwhelmed or if there is persistent bicarbonate loss. So here are the three absolute indications of bicarbonate therapy in metabolic acidosis:

1. Bicarbonate < 6 mEq/dl
2. pH < 7.0
3. Chronic renal failure

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Final Considerations and Take Home Points

When dealing with metabolic acidosis its important to keep in mind that the acidosis can be due to excess acid or due to loss of bicarbonate. To identify which category our patient falls under we need to calculate the anion gap. High anion gap suggests that there is excess acid in the system. Whereas normal anion gap suggests that there is a loss of bicarbonate through renal or GI routes.

Equipped with this knowledge it becomes easy to cater the treatment to our patients. Patients with excess acid don't require bicarbonate supplementation unless the acidosis is severe (pH<7.0 or bicarbonate < 6.0 mEq/dl). In these patients treatment of the underlying pathology will revert the metabolic acidosis. On the other hand, patients who have a renal loss of bicarbonate like those with chronic renal failure, front change chronic diarrhea or renal tubular acidosis will benefit from bicarbonate therapy.

Owned and Published by Dr. Madhusudhan 28, Cathedral Garden Road,Chennai - 34.
Printed by S. Baktha at Dhevi Suganth Printers 52, Jani Batcha Lane, Royapettah, Chennai -14.
Publication on : Final Week of Every Month Posted on 29.11.2023