

Acid-Base Disturbance: A Comprehensive Flowchart-based Diagnostic Approach

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Abstract

Approaching acid-base disturbances is considered a medical problem among healthcare practitioners. Practice-wise, system-based approach should be used to simplify the diagnosis and facilitate management. Flowcharts are considered education tools that can organize thoughts and standardize care. Using a flowchart approach make the practitioners solve any complex acid-base disturbance and facilitate the teaching of such topic.

Keywords: Acid-base; Metabolic; Respiratory; Acidosis; Alkalosis; Anion gap; Osmol gap; Flowchart

Introduction

The acid-base homeostasis is carefully balanced through a delicate series of interactions which involve organs such as the lungs and kidneys, as well as a complex system of buffers. Optimal body function and metabolic systems are kept in check by maintaining a normal pH (7.35-7.45) of arterial blood. Values less than <7.35 are termed acidemia, whereas values more than >7.45 are referred to as alkalemia. Any disorder that lowers the pH to <7.35 is called acidosis, while a disorder that increases the pH >7.45 is called alkalosis [1-3]. The Henderson-Hasselbalch equation describes the Regulation of the systemic pH by means of metabolic and respiratory components [4]:

$$\text{pH} = 6.1 + \log \left(\frac{[\text{HCO}_3^-]}{0.03 \cdot \text{PCO}_2} \right)$$

The equation demonstrates that the pH is determined by bicarbonate (HCO_3^- the metabolic component) and carbon dioxide (PCO_2 the respiratory component) ratio [4,5].

The main categories of acid base disturbances include: respiratory disorders (acidosis and alkalosis) and metabolic disorders (acidosis and alkalosis). Notably, respiratory disorders are expressed primarily as changes in PCO_2 while metabolic disorders are expressed primarily as changes in HCO_3^- [6,7].

Acid-base analysis can be a complicated and time-consuming process, not to mention a confusing topic among practicing physicians and clinical trainees. Diagnostic evaluation of acid-base disturbances, coupled with clinical data, can provide vital information to guide clinicians in making important management decisions in patient care. However if not properly applied, such an important ancillary test can become confusing and hinder health care providers especially in the critically ill.

Flowcharts are considered arbitrary illustrations, also known as a logical illustration, which is well schematized and text-redundant. These types of visual illustrations serve to facilitate the learning process and promote knowledge acquisition [8]. In this article, a simplified flowchart is demonstrated to provide a diagnostic frame-work for healthcare professionals when interpreting acid-base disturbances in the clinical setting and as a tool for medical education in a work-based environment.

Review

The flowchart developed consists of five basic steps. Depending on standard values employed in the hospital at which the healthcare

provider is working, the acid-base result can be simply categorized as low, normal or high (Figure 1).

The 5 steps are as follow:

Step 1: Check the pH

Step 2: Check the PCO_2

Step 3: Check the HCO_3^- (if PCO_2 is normal)

Step 4: Calculate the compensation

Step 5: Calculate the anion gap (AG).

Explanation of the Five Steps

Step 1: Check the pH. If the result is low (<7.35), this means there is acidemia and if high (>7.45), then alkalemia is present. On the contrary, If the pH is normal (7.35-7.45), then the provider should proceed to the next step in order to determine the likelihood of a mixed acid-base disturbance.

Step 2: Check the PCO_2 . If acidemia is present, then a low PCO_2 (<35 mmHg) indicates a metabolic acidosis, while a high PCO_2 (>45 mmHg) indicates respiratory acidosis. On the other hand if alkalemia is present, then a low PCO_2 indicates a respiratory alkalosis, as well as a metabolic alkalosis if PCO_2 is high. Alternatively, if a normal pH with low PCO_2 is encountered, then a mixed respiratory alkalosis and metabolic acidosis is likely. Although, a normal pH with a high PCO_2 means a mixed respiratory acidosis and metabolic alkalosis is present. In the event where the PCO_2 is within normal range (35-45 mmHg) [2], proceed to the next step.

Step 3: If the PCO_2 value is normal, then HCO_3^- should be checked. It is important to mention that even if an abnormal PCO_2 is encountered, the HCO_3^- is still useful in calculating the degree of compensation later on. In other words, checking HCO_3^- at this stage in the presence of a normal PCO_2 value is used to support the diagnosis of either acidemia or alkalemia.

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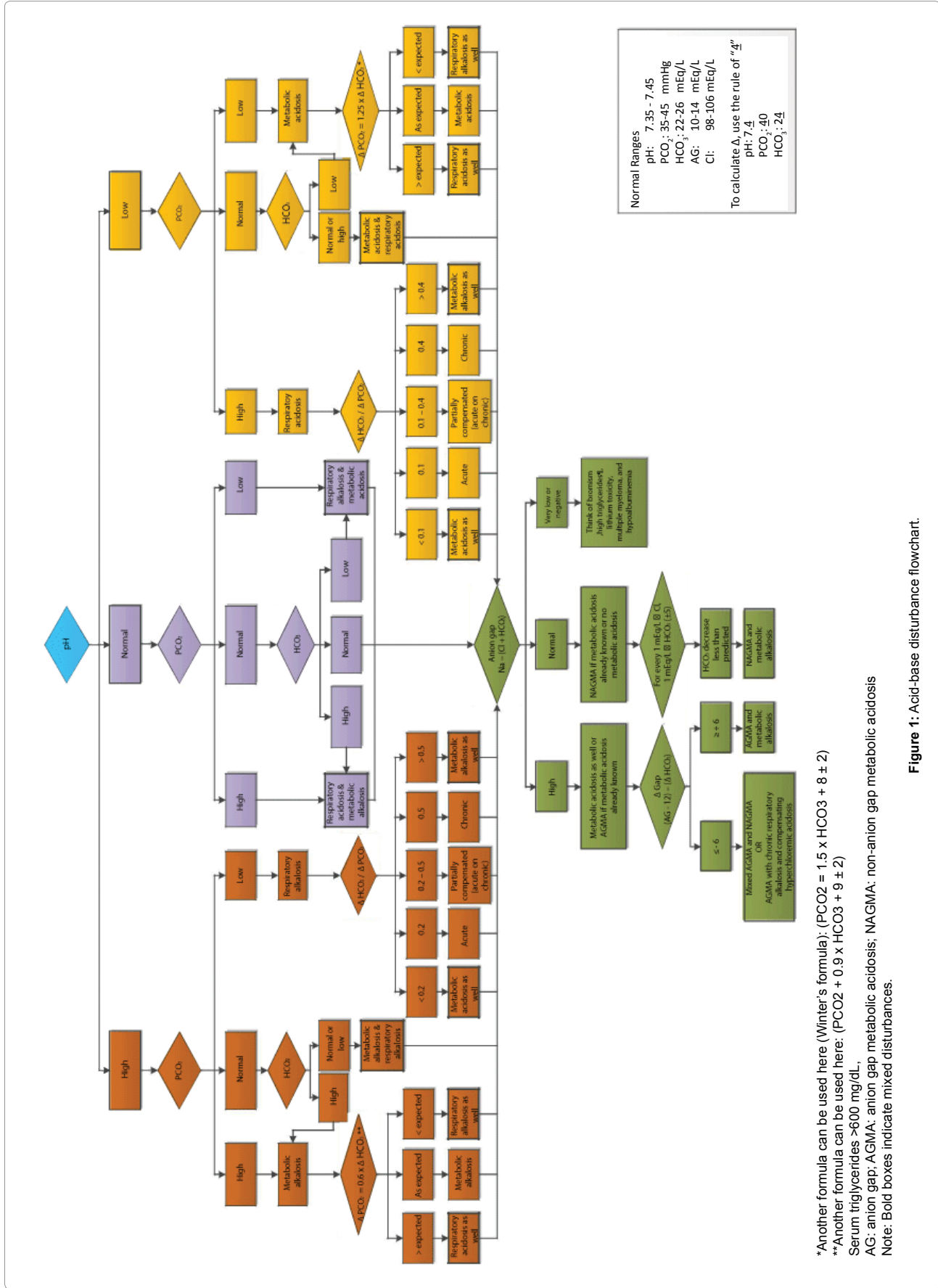


Figure 1: Acid-base disturbance flowchart.

If acidemia with a normal PCO_2 and low HCO_3^- (<22 mEq/L) is present, this would suggest metabolic acidosis. However, if the HCO_3^- is within normal range (22 – 26 mEq/L) [9] or high (>26 mEq/L), then respiratory acidosis and metabolic acidosis are present. Although, if alkalemia with a normal PCO_2 and high HCO_3^- is evident, this situation indicates metabolic alkalosis. Whereas a normal or low HCO_3^- , indicates both respiratory and metabolic alkalosis. However, if the pH and PCO_2 are normal, while the HCO_3^- is low, then a mixed respiratory alkalosis and metabolic acidosis is likely. Alternatively, a normal pH and PCO_2 with a high HCO_3^- would indicate the presence of a mixed respiratory acidosis and metabolic alkalosis. In case of normal values of pH, PCO_2 and HCO_3^- then proceed to step 4 in the flowchart.

Step 4: Calculate the compensation (Table 1).

Metabolic acidosis: The decrease in PCO_2 is approximately equal to 1.25 times the decrease in HCO_3^- [10]. Therefore, the degree of compensation can be calculated using this formula:

$$(\Delta PCO_2 = 1.25 \times \Delta HCO_3^-)$$

Winter's formula ($PCO_2 = 1.5(HCO_3^-) + 8 \pm 2$) can be used to determine the expected degree of compensation as well [11]. If the PCO_2 is more than expected, then respiratory acidosis is likely. While if it is less than expected, then there is respiratory alkalosis as well.

Metabolic alkalosis: The increase in PCO_2 is approximately equal to 0.6 times the increase in HCO_3^- [10]. Therefore, the degree of compensation can be estimated using the following formula:

$$(\Delta PCO_2 = 0.6 \times \Delta HCO_3^-)$$

Alternatively, a different formula can be used ($PCO_2 = 0.9(HCO_3^-) + 9 \pm 2$) to calculate the degree of compensation in the presence of metabolic alkalosis [12].

If the calculated PCO_2 is more than expected, then a respiratory

acidosis is present. While a calculated PCO_2 that is less than expected suggests the presence of a respiratory alkalosis as well.

Respiratory acidosis: This can either be defined as acute or chronic respiratory acidosis. In acute respiratory acidosis (2-3 days), there is 1 mEq/L increase in HCO_3^- for every 10 mmHg increase in PCO_2 , that is, a one to ten ratio (1:10). On the other hand in chronic respiratory acidosis (>3 days), there should be 4 mEq/L increase in HCO_3^- for every 10 mmHg increase in PCO_2 , meaning a four to ten ratio (4:10) [13].

Therefore, if the estimated change in HCO_3^- and PCO_2 ($\Delta HCO_3^-/\Delta PCO_2$) are determined, then three possible conclusions are likely: a value of 0.1 indicates acute respiratory acidosis and a value of 0.4 suggests chronic respiratory acidosis. However, a value between 0.1 – 0.4 would indicate acute on chronic respiratory acidosis.

Alternatively, if the value is <0.1, then metabolic acidosis is present, while if the estimated change is >0.4, then metabolic alkalosis is likely.

Respiratory alkalosis: In a similar manor, this can either be defined as acute or chronic respiratory alkalosis. In acute respiratory alkalosis (2-3 days), there is 2 mEq/L decrease in HCO_3^- for every 10 mmHg decrease in PCO_2 that is, a two to ten ratio (2:10). On the contrary in chronic respiratory acidosis (>3 days), there will be a 5 mEq/L decrease in HCO_3^- for every 10 mmHg decrease in PCO_2 meaning a five to ten ratio (5:10) [12].

Therefore, if the estimated change in HCO_3^- and PCO_2 are determined ($\Delta HCO_3^-/\Delta PCO_2$), then three possible conclusions are likely: a value of 0.2 which indicates acute respiratory alkalosis and a value of 0.5 would suggest chronic respiratory alkalosis. However, a value between 0.2-0.5 suggests an acute on chronic respiratory alkalosis. Alternatively, if the estimated change is <0.2, then there is also a metabolic acidosis, while if the result is >0.5, a metabolic alkalosis exists as well.

Step 5: Calculate the anion gap. This step must be done regardless of the previous results and even if all parameters are normal. The anion gap can be calculated using this formula:

$$Na - (Cl + HCO_3^-)$$

High anion gap (>15 mEq/L): This would suggest the presence of a metabolic acidosis regardless of prior estimations and means an anion gap metabolic acidosis exists if acidosis was already determined in the previous steps.

In this case, the delta gap (Δ gap), or estimated degree of change anticipated in the anion gap should be calculated using this formula:

$$(\Delta G - 12) - (\Delta HCO_3^-)$$

The normal range of the Δ gap is [(-6) – (+6)] [14]. If the Δ gap is ≤ -6 , then this would indicate either one of the following: a mixed AGMA and NAGMA, or AGMA with chronic respiratory alkalosis and compensating hyperchloremic acidosis. However if Δ gap is $\geq +6$, this would mean AGMA with metabolic alkalosis is likely [15].

In any patient with an AGMA, it is necessary to calculate an osmol gap which can help predict potentially life-threatening toxic alcohol ingestion.

The osmol gap can be determined as follows:

$$\text{Osmol gap} = \text{measured osmolality} - \text{calculated osmolality}$$

The calculated osmolality is easily estimated using this formula:

$$\text{Calculated Osmolality} = 2(Na) + \text{Glucose}/18 + \text{BUN}/2.4 + \text{ETOH}/4.6$$

Metabolic acidosis	$\Delta PCO_2 = 1.25 \times \Delta HCO_3^-$
	$PCO_2 = 1.5(HCO_3^-) + 8 \pm 2$
	For AGMA, the rise in AG should be equal to the fall in HCO_3^- .
	For NAGMA (hyperchloremic), the fall in HCO_3^- should be equal to the rise in Cl.
Metabolic alkalosis	Limit of compensation: PCO_2 will not fall below 10–15 mmHg.
	$\Delta PCO_2 = 0.6 \times \Delta HCO_3^-$
	$PCO_2 = 0.9(HCO_3^-) + 9 \pm 2$
Respiratory acidosis	Limit of compensation: PCO_2 rarely exceeds 55 mmHg.
	Acute
	HCO_3^- increases 1 mEq/L (0.25–1.75) for every 10 mmHg increase in PCO_2 .
	pH decreases 0.08 for every 10 mEq/L increase in HCO_3^- .
	Chronic
Respiratory alkalosis	HCO_3^- increases 4 mEq/L for every 10 mmHg increase in PCO_2 (± 4).
	Limit of compensation: HCO_3^- will rarely exceeds 38–45 mEq/L.
	Acute
	HCO_3^- drops 1 to 3.5 mEq/L for every 10 mmHg drop in PCO_2 .
	Limit of compensation: HCO_3^- rarely falls below 18 mEq/L.
Respiratory alkalosis	Chronic
	HCO_3^- drops 2–5 mEq/L for every 10 mmHg drop in PCO_2 .
	Limit of compensation: HCO_3^- is rarely below 12–14 mEq/L.

Table 1: Formulas and relationship between different acid-base disturbances.

When the measured osmolality differs by 10-15 mOsm/kg H₂O from the calculated osmolality, the presence of an unmeasured substance should be considered [1,16]. However, it is important to mention that toxic alcohol ingestion cannot be excluded by a normal osmol gap level and needs to be carefully considered within the context of the patient presentation [17]. Causes of increased osmol gap (MMEEGGLI) are listed below:

- Methanol
- Mannitol
- Ethanol
- Ethylene glycol
- Glycine
- Glycerol
- Lactate
- Isopropyl alcohol

Normal anion gap: If acidosis was determined in the previous steps, then a normal anion gap (10 – 14 mEq/L) [13] suggests normal anion gap metabolic acidosis (NAGMA). Consequently, there should be a 1 mEq/L increase in chloride (above the normal of 100), and 1 mEq/L decrease in HCO₃ (±5). If the decrease in HCO₃ is less than expected, then this would indicate both NAGMA and metabolic alkalosis [10].

Very low or negative anion gap: In this situation, careful consideration of an underlying additional metabolic cause should be examined, namely hypoalbuminemia, as the anion gap is affected by a low albumin level. In other words, with every 1 g/dl decrease in serum albumin, the anion gap will decrease by 2.5 mEq/L [18].

In the end, after going through the steps mentioned above and reviewing the possible causes of each condition (Table 2), the interpretation and subsequent correction of an acid-base problem should always be evaluated in context of the clinical data obtained from the patient's history and physical exam findings [19,20].

Discussion

Although the evaluation of acid-base disturbances can be a daunting task, a simplified and yet organized approach with the clinical presentation in mind can help aid healthcare practitioners in making crucial management decisions that are vital to patient care. The flowchart, as mentioned previously, serves to help those responsible for patient care approach acid-base abnormalities in a more standardized fashion, creating a framework for further management strategies. It is important to mention that many explanations are available which address the issue of acid-base interpretation, but in the proposed flowchart, a more practical approach is emphasized, eliminating unnecessary steps which could hinder the overall evaluation.

As with any flowchart, there are certain restrictions to its use. Conditions in which a patient cannot compensate metabolic acidosis, such as being intubated, can hinder the application of the flowchart. In addition, the values in the flowchart are not fixed and may differ depending on laboratory standard reference values used. Another circumstance in which such a flowchart may not be accurate is in pregnant patients. Differences in values found during pregnancy are considered acceptable physiological changes as pregnant women tend to have a higher pH and lower PCO₂ secondary to normal compensatory measures.

Metabolic acidosis
AGMA (MUDPILERS ACT)
Methanol intoxication
Uremia
Diabetic ketoacidosis
Paraldehyde
Isoniazide
Lactic acidosis
Ethanol
Rhabdomyolysis
Salicylates
Alcoholic ketoacidosis
Cyanide, Carbon monoxide
Toluene
NAGMA
GI bicarbonate loss (diarrhea).
Renal tubular acidosis.
Carbonic anhydrase inhibitors.
Ureteral diversions.
Rapid normal saline rehydration.
Respiratory acidosis
Central nervous system depression
Pleural disease (pneumothorax, pleural effusion)
Lung disease (ARDS, COPD, pulmonary edema, pneumonia)
Airway obstruction
Neuromuscular disorders (Guillain-Barré syndrome, myasthenia gravis)
Thoracic injury (flail chest)
Metabolic alkalosis
Volume contraction (vomiting, gastric suction, diuretics)
Excess glucocorticoids or mineralocorticoids (eg, Cushing's syndrome)
Hypokalemia
Bartter's syndrome
Alkali ingestion/infusion
Post-respiratory acidosis
Respiratory alkalosis
CNS disease (Cerebrovascular accident)
Toxins (Salicylates)
High altitude
Severe anemia
Pregnancy
Lung disease/hypoxia (asthma, pneumonia, pulmonary embolism, pulmonary edema, pulmonary fibrosis)
Anxiety
Cirrhosis of the liver
Septicemia

Table 2: Causes of acid-base disturbances.

Future study in this regard should aim to further validate this conclusion, as well as to address the issue of the use of such a flowchart as an educational tool for educational purposes.

Conclusion

Acid-base disturbances are common problems and can be the result of numerous disease entities. Integrating the patient's clinical data which includes; history and physical examination findings, with a step-wise systematic flowchart approach, can aid healthcare providers in overcoming diagnostic dilemmas and subsequently take appropriate action. In addition, a flowchart-based approach facilitates the learning process and can be a useful teaching tool when addressing complex acid-base disturbances.

Authors' Contributions

Alshehri AA reviewed the literature, designed the flowchart and wrote the

draft manuscript. Alyahya MA reviewed the contents and involved in writing and proof writing the body text of the whole manuscript. Alsolamy SJ revised the whole scientific contents and gave the final approval for the version to be submitted.

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Note: ^aThe application is available for free in the following link: <https://itunes.apple.com/sa/app/abg-test/id887189397?mt=8>

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