Stewart's approach to acid-base disorders Evaluation of Acid-Base Disorders in Two Patients Using Stewart's Approach

Evaluation of Acid-Base Disorders in Two Patients Using Stewart's Approach

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Disturbances of acid-base balance can result from serious cellular and general consequences. Monitoring of blood pH is clinically important to evaluate and understand the physiological condition of a critically ill patient. The traditional approach to acid-base equilibrium based on the Henderson –Hasselbalch equation focuses on changes in the concentration of bicarbonate (HCO_3^-), the partial pressure of carbon dioxide (pCO_2), the dissociation constant and the solubility of CO_2. The Stewart's approach, however, based on the analysis of the complex components of physiologic fluid, such as sodium (Na^+), potassium (K^+), calcium (Ca^{2+}), magnesium (Mg^{2+}), chloride (Cl^-), pCO_2, lactate, phosphorus, and protein, provides better information and a more accurate conceptual view of acid-base mechanism. In this review, we evaluate two cases of acid-base disturbance, one after major surgery, and the other after liver transplantation, using the Stewart's approach, and compare it with the traditional approach.

Key words: ***acid-base balance, strong ion, Henderson–Hasselbalch, Stewart's approach, anion gap, metabolic acidosis, respiratory alkalosis***

Introduction

Acid-base disturbances remain sophisticated problems in clinical practice. The pH is tightly controlled in acid-base physiology by changes in the plasma hydrogen ion concentration [H^+]. The traditional approach to acid–base control is based on the Henderson–Hasselbalch equation[1,2]. An alternative clinical approach to acid-base balance was cited by Peter A. Stewart, Ph.D in 1981[3-5]. In Stewart's approach, the strong ion difference (SID) of dissociated ions, partial pressure of carbon dioxide (pCO_2), and the sum of acids present in the plasma become the major determinant of [H^+] in the plasma[5,6]. This revolutionalized concept is accepted and widely applied in many European hospitals, especially in critical care units, trauma centers and anesthetic centers, since this approach can identify more major acid-base disturbances than the traditional approach.

The Traditional Approach to Altering pH

In traditional acid-base evaluation, the Henderson and Hasselbalch equation is used: pH = pKa + log ([HCO_3^-] / [0.03 pCO_2 mmHg]) where the pKa value at 37°C is 6.1[1,2]. With this equation, metabolic disorders are not clearly quantified since [HCO_3^-] depends on the partial pressure of (pCO_2) *in vivo*. So, the standard base excess (SBE) and standard bicarbonate theories were introduced

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to reveal underlying metabolic disorders[7,8]. The calculated bicarbonate value is adjusted under a pCO_2 of 40 mmHg (5.3 kPa) to achieve the standard bicarbonate value. The SBE represents the amount of base that needs to be added to the blood to get a normal pH at a pCO_2 of 40 mmHg. The more negative the SBE, the more acidic the blood results.

Stewart's Physicochemical Approach to Altering pH

With the Stewart concept, autoionization of water results in H^+ and OH^-, and the water dissociation can be written as:

$H_2O \leftrightarrow H^+ + OH^-$

Water dissociation remains constant, in other words,

$Kw = [H^+] \times [OH^-]$,

Where Kw is the dissociation constant for water[9,10].

If $[H^+]$ increases, $[OH^-]$ decreases by the same amount. Three variables that determine water dissociation, i.e. the pCO_2, SID, and total weak acid concentration (A_{TOT}), become important in determining the pH (Fig. 1)[11].

Our goal in this report is to compare the traditional approach to acid-base equilibrium based on the CO_2 and HCO_3^- buffering system with the Stewart's approach based on the so-called SID and A_{TOT}.

Examples

Case 1

A 72 years old woman had a motor vehicle accident and sustained severe injuries to the right anterior chest and lower abdomen. She had a history of type 2 diabetes mellitus and hypertension for 5 years, chronic obstructive lung disease for 3 years, and a cholecystectomy for gallstones 2 years previously. Her regular medications include

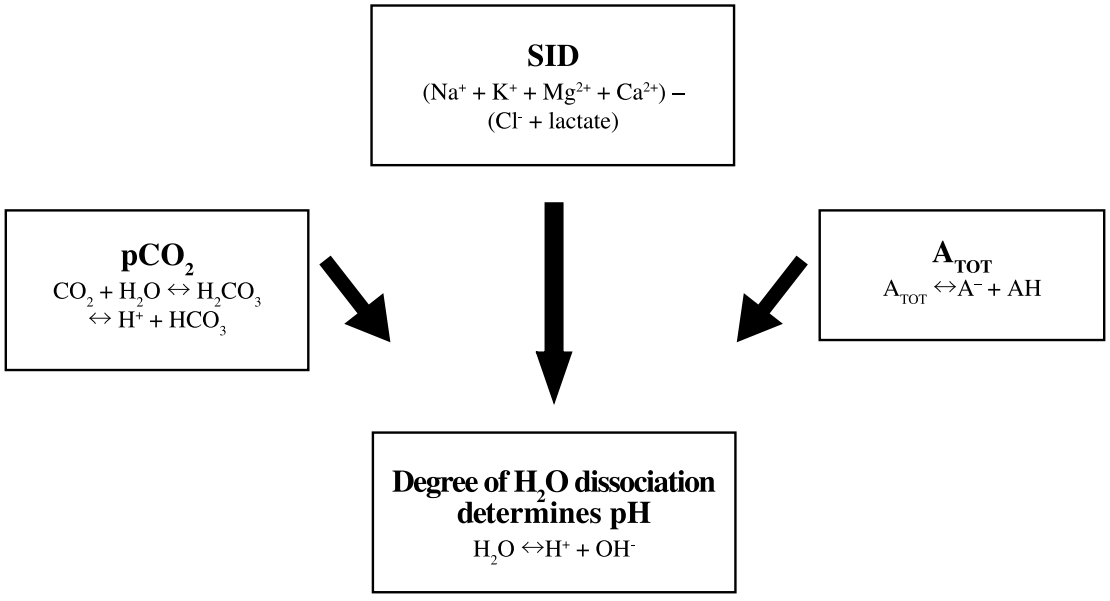

Fig. 1 The three independent factors that determine water dissociation, $[H^+]$ and plasma pH, are: (1) pCO_2; (2) SID; and (3) A_{TOT}. The degree of water dissociation with the release of H^+ is the determinant of body pH. The pCO_2 reflects respiratory disturbances in the pH; SID and A_{TOT} reflects metabolic disturbances in pH. (Abbreviations: A_{TOT}, total weak acid concentration; A^-, dissociated weak acids; AH, associated weak acids; SID, strong ion difference) (Modified from reference 11)

glidiab, nifedipine, and atrovent inhalers prescribed by an endocrinologist in the out-patient department. In the Emergency Room, she was in critical condition with fractures of multiple ribs, pulmonary contusion, and intestinal perforation. Emergency exploratory laparotomy was done and a massive amount of blood was found intraperitoneally. During surgery, large amounts of lactated Ringer's solution and packed red blood cells were given intravenously. Arterial blood gases obtained in the operating room revealed the pH, P_aCO_2, and SBE were 7.10, 30 mmHg, and -19 mEq/l, respectively. The plasma lactate concentration was 11.5 mEq/l after additional blood products and 120 mmol of $NaHCO_3$ were given for resuscitation. Here, the patient had lactic acidosis secondary to blood loss and tissue destruction (SBE of -19 mEq/l, increased lactic acid) with acute respiratory acidosis (calculated pCO_2 of 21 mmHg, less than the actual P_aCO_2 of 30 mmHg) from poor ventilation because of rib fractures and pain.

After transfer to the intensive care unit (ICU), ventilation was supported with a mechanical ventilator. Her blood gas analysis revealed pH was 7.35, pCO_2 35 mmHg, and SBE -5 mEq/l, and the serum lactate was 8.0 mEq/l. Because her hematocrit was low at 29%, she received 4 units of packed red blood cells intravenously. Six hours later, blood gas analysis showed that her blood pH and SBE had increased to 7.58 and +11 mEq/l, respectively, and pCO_2 and [lactate] had decreased to 34mmHg and 2.1 mEq/l, respectively. Here, the patient had primary metabolic alkalosis due to increased SBE and mild respiratory alkalosis with a pCO_2 of 34 mmHg. The components of the metabolic alkalosis were a combination of lactate clearance, massive blood transfusion (citrate), and $NaHCO_3$ administration. The ventilator settings after adjustment to compensate for metabolic acidosis contributed to the respiratory alkalosis. So, the minute ventilation was reduced to allow mild hypercapnea to normalize the pH to 7.40.

On postoperative day 3 (POD3), the patient had fever and hypotension. An arterial blood gas analysis revealed a pH of 7.31, SBE of -9 mEq/l and arterial lactate of 5.9 mEq/l. The calculated anion gap (AG) was 17 mEq/l, and concentrations of plasma phosphate and albumin were in the normal range. Owing to low central venous pressure and hypotension, she received 8 liters of normal saline and a norepinephrine infusion over the next 24 hrs. Her urine output was only 200 cc despite resuscitation. On post-operative day 4 (POD 4), her arterial blood gas analysis showed a pH of 7.22, pCO_2 of 30 mm Hg, $[HCO_3^-]$ of 12 mEq/l, and SBE of -13 mEq/l, and the arterial lactate was 4.2 mEq/l. There was metabolic acidosis despite the reduction in plasma lactate levels. This was because of the large volume of normal saline infused on POD3, indicating the classic situation of hyperchloremic metabolic acidosis from resuscitation fluids.

On POD5, the patient received 3.5 liters of fluid supplemention with lactated Ringers (Na^+: 130 mEq/l, lactate: 28 mEq/l). The urine output increased and her acid-base status improved with decreased $[Cl^-]$ in the plasma. The patient's blood gas and biochemistry data are summarized in Table 1.

Traditional approach

In this case, the traditional approach indicated that hyperchloremic acidosis should be treated with sodium bicarbonate solution: $NaHCO_3^- \leftrightarrow Na^+ + HCO_3^-$ or Tris-hydroxymethyl aminomethane (THAM): $R\text{-}NH_2 + H_2O + CO_2 \leftrightarrow R\text{-}NH_3^+ + HCO_3^-$. Here, $R\text{-}NH_2$ is THAM and $R\text{-}NH_3^+$ is the protonated form of THAM. Both treatments can donate bicarbonate, cause an increase in plasma $[HCO_3^-]$ and restore the pH to normal.

Application of Stewart's strong ion concept

The traditional approach does not explain how and why the hyperchloremic metabolic acidosis occurs from large amounts of infused normal saline.

Table 1 Case 1 patient's blood gas and biochemistry data

Blood chemistries	On Arrival ICU	7am POD3	8am POD4	6am POD5
Na (mEq/l)	140	139	142	139
K (mEq/l)	3.9	3.2	3.5	3.9
Mg (mEq/l)	1.65	-	1.64	-
Cl (mEq/l)	102	103	114	108
HCO_3 (mEq/l)	18.8	22.1	12	22
pH	7.35	7.31	7.22	7.36
$[H^+]$ (nmol/l)	[44.67]	[48.98]	[60.2]	[43.65]
pCO_2 (mmHg)	35	45.1	30.1	40.0
SBE (mEq/l)	-5	-9	-13	-5
Lactate (mEq/l)	8	5.9	4.2	2.1
Albumin (g/dl)	4.1	-	4.0	-
Phosphate (mg/dl)	3.8	-	3.9	-
Anion Gap (mEq/l)	23.1	17.1	19.5	12.9
SID (mEq/l)	40.6	40.0	34.0	39.5

- SID= { $[Na^+]$ + $[K^+]$ + $[Ca^{2+}]$ + $[Mg^{2+}]$ } - { $[Cl^-]$ + [lactate] }
- Conversion of lactate in mg/dl to mEq/l (mmol/l) involves multiplying by 0.111

Stewart's approach explains the mechanism of hyperchloremic acidosis and its rational treatment. This is important since normal saline is widely used nowadays as a first line resuscitative fluid in most hospitals and may deteriorate underlying acidosis if it is unnoticed.

The Stewart concept starts with strong ions, completely dissociated ions which exist only as charged forms at physiologic pH in biologic solutions. In plasma, strong ions may be inorganic, e.g. Na^+, Cl^-, K^+ or organic, e.g. lactate, where Na^+ and Cl^- are the major and most abundant strong ions.

Strong ion difference

SID = (the sum of all measurable strong cation concentrations in the plasma) minus (the sum of all measurable strong anion concentrations in the plasma)[11]. SID= { $[Na^+]$ + $[K^+]$ + $[Ca^{2+}]$ + $[Mg^{2+}]$ } - { $[Cl^-]$ + [lactate] }

The normal plasma SID is 40 to 42 mmol/l of a net positive charge. Plasma SID changes have a significant influence on water dissociation via the laws of electrical neutrality and mass conservation. A net increase in the strong cation concentration in the plasma will increase the SID. The increase in the SID level decreases H^+ release from water, and thus reduces plasma $[H^+]$ and elevates pH[1,2]. Similarly, a decreased serum SID from strong anion elevation may increase water dissociation with a resultant increase in $[H^+]$ and lower pH.

Normal human plasma is on the alkaline side of neutral with a positive SID. Plasma $[Na^+]$ and $[Cl^-]$ are in the ranges of 135-155 mEq/l and 95-105 mEq/l, respectively. Normal saline solution with no other strong ions present has a SID of 0 mEq/l ($[Na^+]=[Cl^-]=154$ mEq/l). When normal saline is infused intravenously, a net increase in Cl^- anion compared with Na^+ cation results in a decreased SID that causes dissociation of H^+ from H_2O. An increase in plasma $[H^+]$ and a fall in pH could be expected with normal saline infusion. Here, hyperchloremia was the cause of metabolic acidosis. The more negative ions of Cl^- infused, the more acidic the plasma becomes. The rate and dose of normal saline infusion is the main determinant of hyperchloremic acidosis.

The specific treatment for hyperchloremic

acidosis is to increase the SID by adding strong cation to compensate for water dissociation and hydrogen ion release. A solution with a strong cation concentration exceeding the strong anion concentration by 40-42 mEq/l can be given for this purpose since the normal plasma SID is 40-42 mEq/l. Both 130 mmol of sodium bicarbonate (e.g. 7.5% sodium bicarbonate; Na^+ without Cl^-, effective SID of 130 mEq) and 128 mmol of THAM have a high effective SID and can correct this type of acidosis. Therefore, the strong ion approach explains how and why sodium bicarbonate and THAM are used to treat hyperchloremic acidosis. On the contrary, if critically ill patients present with severe hyperlactatemia associated with hypochloremic alkalosis, the laboratory data may show normal values for the pH, [HCO_3^-] and SBE[12].

Case 2

A 58 years old man had liver transplantation. Because of allograft function impairment, the lactate concentration increased to 16 mEq/l 5 hours after transplantation. At that time, arterial blood gas analysis revealed a pH of 7.18, pCO_2 of 30 mmHg, and SBE of -17 mEq/l, indicating metabolic acidosis secondary to lactic acid accumulation with respiratory acidosis due to inadequate postoperative respiration. Measurements of the pulmonary capillary wedge pressure, right ventricular end-diastolic volume, and serum albumin were 12 mmHg, 118 ml, and 2.2 g/dl, respectively. Here, the increase in plasma lactate and decrease in SBE were found simultaneously and primary lactic acidosis was easily defined. We adjusted the mechanical ventilator in an effort to maintain the P_aCO_2 at a level of 26-28 mmHg. Additional fluid supplements were given to reduce the anaerobic metabolism. A total of 50 gm albumin (200 ml of 25% albumin) was administered intravenously. Normal saline resuscitation was not done since it might have increased the metabolic acidosis.

Six hours later, the patient's urine output was reduced and the serum [Na^+] and [Cl^-] were 132 and 104 mEq/l, respectively. Arterial blood gas data showed a pH of 7.32, pCO_2 of 25 mmHg, and SBE of -12 mEq/l. The lactate was still 16 mEq/l. Therefore, an infusion of 100 mEq of 7.5% $NaHCO_3$ was given. Twelve hours later, the patient awoke. The liver function resumed with bile production, and the urine output increased. Arterial blood gas analysis revealed a pH of 7.40, pCO_2 of 35 mmHg and SBE of -1 mEq/ l. The blood lactate decreased by 6 mEq/l to 10 mEq/l. The serum [Na^+] and [Cl^-] were 134 and 101 mEq/l, respectively, showing an increase of [Na^+] by 2 mM and a decrease of [Cl^-] by 3 mM. Although these changes were small, they resulted in a significant increase in the SID from 21 mEq/l to 32 mEq/l. The reduction of serum [Cl^-] occurred because of preserved renal excretion and/or other inter-compartmental shifts. The serum [Na^+] was increased by exogenous Na^+ administration from the $NaHCO_3$ and 25% albumin infusion.

An overview of the patient's data, showed that the SID was low at 32 mEq/l which might be attributed to a low level of A_{TOT} (albumin is 2.7 g/dl, phosphate is 2.9 mg/dl). When the [lactate] then decreased, the SID increased to nearly 40 mEq/l and with pending metabolic alkalosis. Thus the minute ventilation needed to be reduced. The kidneys retained Cl^- over the next few hours and restored the SID to the baseline. When the transplanted liver started to produce albumin, the A_{TOT} increased gradually and reached a new steady SID. The patient's blood gas and biochemistry data 6 hrs and 12 hrs after albumin infusion are summarized in Table 2.

Traditional approach

According to the Henderson-Hasselbalch equation: $pH = pKa \times \log[HCO_3^-/(0.03 \times (pCO_2)]$[1,2]. Changes in plasma HCO_3^- lead to metabolic or non-respiratory acid-base anomalies. Respiratory

compensation leads to changes in pCO_2 to compensate for primary metabolic disorders. Since HCO_3^- and pCO_2 are interdependent, this formula can't be used to explain the complex metabolic acid-base disturbances noted on clinical grounds, especially in critically ill patients. So, an alternate approach to quantify the metabolic component was suggested by Siggard-Anderson and colleagues. The SBE was calculated by the Van Slyke equation[7,8]. The correlation between changes in the SBE and pCO_2 during metabolic acid-base disturbances is shown in Table 3.

The major drawback of the SBE approach is that weak acids, such as plasma proteins and other unmeasured anions, are not included. Most patients in critical units have reduced proteins and increased metabolic anions. Emit and Narins developed the anion gap (AG) approach to determine metabolic disorders[13]. The law of electro-neutrality is applied in this approach.

The sum of cations = The sum of anions (electrical neutrality)

$Na^+ + K^+$ + unmeasured cations = $Cl^- + HCO_3^-$ + unmeasured anions

Anion gap = $(Na^+ + K^+) - (Cl^- + HCO_3^-)$ = 10–12 mmol/litre.

In most critically ill patients, hypoalbuminemia is encountered frequently. For hypoalbuminemic and hypophosphatemic patients, the corrected anion gap = $[(Na^+ + K^+) - (Cl^- + HCO_3^-)]$ – [(0.2 × albumin g/l + 1.5 × phosphate mmol/l)][14]. In patients with severe hyperlactatemia, the corrected anion gap = $\{[(Na^+ + K^+) - (Cl^- + HCO_3^-)] + 0.25 \times (40 - [\text{albumin (g/l)}]) - \text{lactate}\}$[15]. The drawback of this approach is that this equation involves $[HCO_3^-]$. The SBE and AG approach is inadequate since $[HCO_3^-]$ can be changed accordingly with respiratory disturbances, e.g. in hyperventilation.

Application of Stewart's weak acid (A_{TOT}) concept

Stewart's approach includes the non-volatile weak acids (A_{TOT}) as a major component in acid-base disturbances. Plasma A_{TOT} includes inorganic phosphate, albumin and other plasma proteins.

1. Proteins ($[PrTot] = [Pr^-] + [HPr]$)
2. Phosphates ($[PiTot] = [PO_4^{-3}] + [HPO_4^{-2}] + [H_2PO_4^-] + [H_3PO_4]$)

Albumin acts as a weak acid in plasma

Table 2 Case 2 patient's blood gas and biochemistry data

Blood chemistries	6 hrs later	12 hrs later (6 hrs after HCO_3^- infusion)
Na (mEq/l)	132	134
K(mEq/l)	3.8	3.6
Cl (mEq/l)	104	101
Lactate (mEq/l)	16	10
Phosphate (mg/dl)	3.8	2.9
Albumin (g/dl)	2.6	2.7
pH	7.32	7.40
$[H^+]$ (nmol/l)	[47.9]	[39.8]
pCO_2 (mmHg)	25	35
HCO_3^- (mEq/l)	12.5	21
SBE (mEq/l)	-12	-1
Anion gap (mEq/l)	19.3	15.6
Corrected anion gap (mEq/l)	17.0	13.7
SID (mEq/l)	21.45	32.25

Phosphate in mg/dL is converted to mmol/L by multiplying by 0.323

Anion gap = $(Na^+ + K^+) - (Cl^- + HCO_3^-)$

Corrected anion gap = $[(Na^+ + K^+) - (Cl^- + HCO_3^-)] - [(0.2 \times \text{albumin g/l} + 1.5 \times \text{phosphate mmol/l})]$

Table 3 The correlation between changes in SBE and pCO_2 in acid-base disturbances

Acid-base disturbance	pCO_2 (mmHg)	SBE (mEq/L)
Acute respiratory acidosis	>45	= 0
Acute respiratory alkalosis	<35	= 0
Chronic respiratory acidosis	>45	$=0.4 \times (pCO_2-40)$
Chronic respiratory alkalosis	<35	$=0.4 \times (pCO_2-40)$
Metabolic acidosis	$= (1.5\times HCO_3^-)+8$, or = 40+SBE	< -5
Metabolic alkalosis	$= (0.7\times HCO_3^-)+21$, or = 40+(0.6×SBE)	> +5

(Modified from reference 16)

proteins which greatly determines the pH and it's increase might cause acidosis. Thus, in hypoalbuminemic patients, a normal plasma pH and anion gap might be seen despite underlying severe acidosis. In other words, hypoalbuminemia may mask severe metabolic acidosis. This concept is clinically very important since most critically ill patients presenting with metabolic acidosis are in a hypoalbuminemic condition. If this condition is not taken into account, the underlying metabolic acidosis will be missed and uncorrected[17-19]. Normally serum phosphate levels are so low that a change does not affect acid-base disorders much. However, in renal failure patients, hyperphosphatemia could result in acidemia.

Previous studies found that after adjusting for hypoalbuminemia (e.g. corrected anion gap), both the traditional and Stewart approach are similarly reliable in determining acid-base disorders[18,20]. A prospective study in 2007 by Boniatti and colleagues found that Stewart's approach can identify more major acid-base disturbances than the traditional approach[21].

Conclusion

Acid-base disorders are still a great problem in clinical practice, especially in emergency and intensive care units. Many proposed theories for those disturbances have arisen over the years. The Stewart approach may be more accurate than other concepts in determining acid-base disturbances. This "new approach" helps us understand the underlying mechanisms of hyperchloremic acidosis, hyperalbuminemic acidosis, dilution acidosis, contraction alkalosis, and renal tubular acidosis, leading to appropriate treatment.

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以斯圖爾特方式著手處置臨床酸鹼問題

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酸鹼平衡的異常可能起因於嚴重細胞或全身性代謝問題引起。監控血液pH值對 臨床重症病患的評估與處置以及了解其潛在之病生理極其重要。傳統著手處理酸鹼平衡異常，多依循亨德森-黑索巴克公式(Henderson-Hasselbalch equation)原理處置，其主要著眼於血中重碳酸(HCO_3^-)濃度的改變、二氧化碳的分壓(pCO_2)以及二氧化碳(CO_2)的溶解度這三方面。然而斯圖爾特處置(Stewart's approach)是基於分析血液內複雜的各種陰陽離子(強離子)及各種弱酸性蛋白質、磷酸、乳酸等對水釋放氫離子的影響，以期了解更精確及完整的酸鹼失衡機制及原理。在這篇回顧裡，我們經由兩個酸鹼失衡病例的介紹，讓讀者了解由傳統著手處理與由斯圖爾特方式處置的差異。

關鍵詞：酸鹼平衡，強離子，亨德森-黑索巴克，斯圖爾特，陰離子差，代謝性酸中毒，呼吸性鹼毒症

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