EASY WAY TO UNDERSTAND STEWART’S ACID-BASE

FROM “SALINE” TO MORE “PHYSIOLOGIC” FLUID

Yohanes WH George, MD
EASY WAY TO UNDERSTAND STEWART’S ACID-BASE

Yohanes WH George, MD
NOTICE

Medicine is an ever-changing field. Because of new research and clinical experience broaden our knowledge, changes in treatment and drug therapy may become necessary or appropriate, Readers are advised to check the most current product information provided by the manufacturer of each drug to be administered to verify the recommended standard of administration. It is the responsibility of the licensed prescriber, relying on experience and knowledge of the patient, to determine the best treatment of each individual patient. Neither the publisher nor the author assume any liability for any injury and/or damage to persons or property arising from this publication.
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Dedication

To my great teacher and mentor;

In memoriam

DR. Iqbal Mustafa, MD. FCCM
the pioneer of the modern critical care medicine in Indonesia,
Head of Intensive Care Unit Harapan Kita Hospital (1992-2004),
Jakarta- Indonesia
In critical care and anesthesia medicine, fluid administration is a key element of resuscitation. Currently, there are still controversies regarding fluid resuscitation strategies, both on ‘balanced fluid’ strategy, known as ‘goal-directed therapy’, and from ‘fluid option’ point of view, which is about fluid type selection. In terms of ‘fluid option’, controversial debate about crystalloid and colloid has lasted for a long time and is no more a special concern. Selection of resuscitation fluids based on their effects on acid-base balance of the body is currently a particular concern. Evidences suggest that saline use in fluid resuscitation causes hyperchloremic acidosis, therefore nonsaline-based fluid, also known as ‘balanced fluid’, is currently invented to avoid acidosis effect.

The mechanism of acidosis following saline administration is based on acid-base balance method by Stewart, that is also called quantitative method or physicochemical approach. Unfortunately, this theory is not widely understood despite the fact that it has been known for quite some time (since 1978) and is being accepted slowly in critical care and anesthesia medicine, which is partly caused by its complexity and being not easily understood.

The Department of Anesthesia of RSCM - FKUI finds that this handbook of “EASY WAY TO UNDERSTAND STEWART’S ACID-BASE” is very useful and it will hopefully simplify the understanding of acid-base balance disturbance mechanism based on Stewart’s method for doctors, especially anesthesiologists and doctors who work in emergency departments and critical care units, which will eventually improve the safety and quality of resuscitation fluids selection. We send our special thanks to dr. Yohanes WH George who made this handbook schematic, practical and easy to understand.

Aries Perdana, MD.
Head of Department of Anesthesiology and Intensive Care Unit
Cipto Mangunkusumo Hospital, Medical Faculty, University of Indonesia
Preface

Understanding the chemistry of water and hydrogen ions is an important part of understanding the living system because hydrogen ions participate in so many reactions. One interesting facet of human homeostasis is the tight control of hydrogen ion concentration, [H+]. As metabolism creates about 300 liters of carbon dioxide each day, and as we also consume about several hundred mEq of strong acids and bases in the same period, it is remarkable that the biochemical and feedback mechanism can maintain [H+] between 30 and 150 nanoEq/liter.

Appreciation of the physics and chemistry involved in the regulatory process is essential for all life scientists, especially physiologists. Many physiology textbooks start the discussion of acid-base equilibrium by defining pH, which immediately followed by the Henderson-Hasselbalch equation.

Attention has recently shifted to a quantitative physicochemical approach to acid-base physiology. Many of the generally accepted concepts of hydrogen ion behaviour are viewed differently. This analysis, introduced by Peter Stewart in 1978, provides a chemical insight into the complex chemical equilibrium system known as acid-base balance.

The impact of Stewart’s analysis has been slow, but there has been a recent resurgence in interest, particularly as this approach provides explanations for several areas which are otherwise difficult to understand (e.g. dilutional acidosis, acid-base disorders related to changes in plasma albumin concentration).

Undoubtedly, the physicochemical approach will become more important in the future and this brief review provides an introduction to this method.

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Pages https://www.facebook.com/critcaremedcom
STEWART’S APPROACH IN BRIEF

• GENERAL PRINCIPLES OF STEWART’S APPROACH
  ‣ **Electroneutrality.** In aqueous solutions in any compartment, the sum of all the positively charged ions must equal to the sum of all the negatively charged ions.
  ‣ **The dissociation equilibria** of all incompletely dissociated substances, as derived from the law of mass action, must be satisfied at all times.
  ‣ **Conservation of mass,** the amount of a substance remains constant unless it is added, removed, generated or destroyed. The relevance is that the total concentration of an incompletely dissociated substance is the sum of concentrations of its dissociated and undissociated forms.

Stewart PA. How to understand acid-base. A quantitative acid-base primer for biology and medicine. Elsevier 1981
Mathematical analysis

- The physico-chemical acid-base approach (Stewart’s approach) is different from the conventional approach based on the Henderson-Hasselbalch equation, and requires a new way of approaching acid-base problems.
- In Stewart approach, the \([H^+]\) is determined by the composition of electrolytes and PCO2 of the solution.
- Mathematical analysis shows that it is not absolute concentrations of almost totally dissociated (“strong”) ions that influence hydrogen ion concentration, but the difference between the activities of these strong ions (This “strong ion difference” is commonly abbreviated “[SID]”).

STRICTLY ION DIFFERENCE

- **DEFINITION:**
  - The strong ion difference is the charge imbalance of the strong ions. In detail, the strong ion difference is the sum of the concentration of the strong base cations, less the sum of the concentrations of the strong acid anions.
  - Strong electrolytes are those which are fully dissociated in aqueous solution, such as the cation sodium (Na\(^+\)), or the anion chloride (Cl\(^-\)). **BECAUSE STRONG IONS ARE ALWAYS DISSOCIATED, THEY DO NOT PARTICIPATE IN CHEMICAL REACTIONS (UNMETABOLIZABLE IONS).** Their only role in acid-base chemistry is through the ELECTRONEUTRALITY relationship
THE GAMBLEGRAM

STRONG ION DIFFERENCE IN WATER

Water dissociation into $[H^+]$ and $[OH^-]$ determined by change in [SID]

The $[H^+] = 4.0 \times 10^{-8}$ Eq/L (very small)

$[Na^+] + [K^+] - [Cl^-] = [SID]$

$140 + 4 - 102 = 34$ mEq/L

$[H^+]$ $\uparrow \uparrow$

Acidosis

$[OH^-]$ $\uparrow \uparrow$

Alkalosis

THE RELATIONSHIP BETWEEN [SID] AND pH/$[H^+]$
1. Virtually all solutions in human biology contain water and aqueous solutions provide a virtually inexhaustible source of \([H^+]\).

2. In these solutions, \([H^+]\) concentration is determined by the dissociation of water into \(H^+\) and \(OH^-\) ions.

3. Changes in \([H^+]\) concentration or \(pH\) occur **NOT** as a result of how much \([H^+]\) is added or removed **BUT** as a consequence of water dissociation in response to change in \([SID]\), \(PCO_2\) and weak acid.
**EASY WAY TO UNDERSTAND STEWART'S ACID-BASE**

**STRONG ION GAP (SIG)**

**SIDa** = \([Na^+] + [K^+] + [Mg^{++}] + [Ca^{++}] - [Cl^-] - [Lactate^-]\)

**SIDe** = 12.2 × \(pCO_2/(10-pH)+10 \times [alb] \times (0.123 \times pH-0.631) + [PO_4^{-}] \times (0.309 \times pH-0.469)\)

**SIG = SIDa − SIDe** → Normal value = zero


**pH or [H+] DETERMINED BY**

**TWO VARIABLES**

**INDEPENDENT VARIABLE**

Determine

**DEPENDENT VARIABLE**

*Primary (cause)*

*Secondary (effect)*
INDEPENDENT VARIABLES

- CO₂
- pCO₂
- STRONG ION DIFFERENCE
- WEAK ACID
- Aₜot

Controlled by the respiratory system
The electrolyte composition of the blood (controlled by the kidney)
Weak Acid, The protein concentration (controlled by the liver and metabolic state)

EVERY CHANGE OF THESE VARIABLE WILL CHANGE THE pH

DEPENDENT VARIABLES

- H⁺
- OH⁻
- CO₃²⁻
- A⁻
- HCO₃⁻

IF THESE VARIABLE CHANGE, THE INDEPENDENT VARIABLES MUST HAVE CHANGED
THE PRACTICAL POINT

Henderson-Hasselbalch

- pH
- Respiratory
- Metabolic
- $P_{CO_2}$
- Base Excess-HCO$_3^-$

Stewart’s Approach

- pH
- Respiratory
- Metabolic
- $P_{CO_2}$
- [SID]
- $A_{tot}$

THE DIFFERENCE

Determinants of plasma pH, as assessed by the H-H. Base excess and standard HCO$_3^-$ determine the metabolic component of plasma pH.

Determinants of plasma pH, at 37°C, as assessed by the Strong Ion Difference [SID] model of Stewart. [SID$^+$] and [$A_{tot}$] determine the metabolic component of plasma pH.
THE DIFFERENCE

- The Stewart approach emphasizes mathematically independent and dependent variables.
- Actually, HCO₃⁻ and H⁺ ions represent the effects rather than the causes of acid-base derangements.

CLASSIFICATION OF PRIMARY ACID BASE DISTURBANCE


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**RESPIRATORY**

- Abnormal pCO₂
  - Hypocarbia: Respiratory alkalosis
  - Hypercarbia: Respiratory acidosis

**ACIDOSIS**

**ALKALOSIS**

**Metabolic**

- Abnormal Strong Ion Difference
  - Water
  - Abnormal Strong Anion
    - Chloride
    - Unmeasured Anion
      - Deficit
      - Excess
    - Hyperchloremia
      - Hyperchloremic acidosis
    - Hypochloremia
      - Hyperchloremic alkalosis
  - Positive
  - Lactic / keto acidosis

- Abnormal Weak acid
  - Alb
  - Po₄⁻
  - Hyperchloremic acidosis
  - Hyperchloremic alkalosis
  - Hyperalbuminemia
    - Hyperalbuminemic acidosis
  - Hyperparathyroidism
    - Hyperparathyroidic acidosis

**Liver and Kidney**

"Modified George 2015"
**WATER DEFICIT**

\[
\begin{align*}
\text{Na}^+ &= 140 \text{ mEq/L} \\
\text{Cl}^- &= 102 \text{ mEq/L} \\
[SID] &= 38 \text{ mEq/L}
\end{align*}
\]

\[
\begin{align*}
140/1/2 &= 280 \text{ mEq/L} \\
102/1/2 &= 204 \text{ mEq/L} \\
[SID] &= 76 \text{ mEq/L}
\end{align*}
\]

\[
[SID] : 38 \rightarrow 76 = \text{alkalosis}
\]

**CONTRACTION ALKALOSIS**

**WATER EXCESS**

\[
\begin{align*}
\text{Na}^+ &= 140 \text{ mEq/L} \\
\text{Cl}^- &= 102 \text{ mEq/L} \\
[SID] &= 38 \text{ mEq/L}
\end{align*}
\]

\[
\begin{align*}
140/2 &= 70 \text{ mEq/L} \\
102/2 &= 51 \text{ mEq/L} \\
[SID] &= 19 \text{ mEq/L}
\end{align*}
\]

\[
[SID] : 38 \rightarrow 19 = \text{Acidosis}
\]

**DILUTIONAL ACIDOSIS**
### ABNORMAL IN SID AND WEAK ACID

<table>
<thead>
<tr>
<th>[SID]</th>
<th>[SID]↓</th>
<th>[SID]↑↑</th>
<th>[SID]↓↓</th>
<th>[SID]↓↓</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cl</td>
<td>Cl ↑</td>
<td>Cl ↓</td>
<td>Cl ↓</td>
<td>Cl ↓</td>
</tr>
<tr>
<td>Alb</td>
<td>Alb</td>
<td>Alb</td>
<td>Alb</td>
<td>Alb</td>
</tr>
<tr>
<td>PO₄</td>
<td>PO₄</td>
<td>PO₄</td>
<td>PO₄</td>
<td>PO₄</td>
</tr>
</tbody>
</table>

**THE EFFECT OF SALINE AND BALANCED FLUID FROM THE STEWART’S PERSPECTIVE**

Stewart’s approach not only explains fluid induced acid–base phenomena but also provides a framework for the design of fluids for specific acid–base effects.
How does saline cause acidosis?

**PLASMA + Saline 0.9%**

Plasma:
- $Na^+ = 140 \text{ mEq/L}$
- $Cl^- = 102 \text{ mEq/L}$
- $[SID] = 38 \text{ mEq/L}$

NaCl 0.9%:
- $Na^+ = 154 \text{ mEq/L}$
- $Cl^- = 154 \text{ mEq/L}$
- $[SID] = 0 \text{ mEq/L}$

$[SID] : 38 \rightarrow \text{normal pH}$

SALINE CAUSE ACIDOSIS BY DECREASING $[SID]$ DUE TO THE HYPERCHLOREMIC

Plasma:
- $Na^+ = (140 + 154)/2 \text{ L} = 147 \text{ L}$
- $Cl^- = (102 + 154)/2 \text{ L} = 128 \text{ L}$
- $[SID] = 19 \text{ L}$

$[SID] : 19 \downarrow \rightarrow \text{pH} \downarrow \rightarrow \text{More acidosis}$
THE DIFFERENCES BETWEEN H-H AND STEWART’S APPROACH IN EXPLAINING ACIDOSIS FOLLOWING INFUSION OF SALINE

Increase in [Cl⁻] > [Na⁺]

Strong Ion Difference falls

Water dissociates

D.A Story, Critical Care and Resuscitation 1999; 1:151-156

simple analogy

PLASMA + LACTATE RINGER

Plasma

Na⁺ = 140 mEq/L
Cl⁻ = 102 mEq/L
[SID]= 38 mEq/L

Lactate ringer

Cation⁺ = 137 mEq/L
Cl⁻ = 109 mEq/L
Lactate = 28 mEq/L
[SID] = 0 mEq/L

1 liter + 1 liter

WHAT HAPPEN WHEN WE GIVE LR TO THE PLASMA

[SID] : 38
**EASY WAY TO UNDERSTAND STEWART’S ACID-BASE**

- TBW 50 kg.
- TBW 30% = 0.65 kg = 30L
- [Na⁺] = 140 = 30.140 = 4200
- [Cl⁻] = 100 = 30.100 = 3000

**Give 2 liters of LR:**
- [Na⁺] = 137 x 2 L = 274
- [Cl⁻] = 109 x 2 L = 218

**Systemic [SID] (30+2L):**
- [Na⁺] = 4508/32 = 140.8
- [Cl⁻] = 3308/32 = 103.3
- [SID] = 37.0 (more acidosis)

**Give 2 liters of 0.9% Sodium Chloride:**
- [Na⁺] = 154 x 2 L = 308
- [Cl⁻] = 154 x 2 L = 308

**Systemic [SID] (30+2L):**
- [Na⁺] = 4474/32 = 139.8
- [Cl⁻] = 3218/32 = 100.5
- [SID] = 39.3 (more alkalosis)

**SALINE INFUSION CAUSE MORE ACIDOSIS THAN LACTATE RINGER**

**Simple analogy**

- [SID] close to normal after lactate ringer infusion

- 2 liters

- [SID] become 34 → plasma pH become more alkalosis than plasma pH after saline infusion

George 2015
LARGE INFUSION SALINE CAUSE MORE ACIDOSIS

Give 10 liters of 0.9% Sodium Chloride:
\[ [Na^+] = 154 \times 10 \text{L} = 1540 \]
\[ [Cl^-] = 154 \times 10 \text{L} = 1540 \]
\[ \text{Dilutional } [\text{SID}] = 30 + 10 \text{L} = 30.0 \text{(dilutional acidosis)} \]

Give 2 liters of 0.9% Sodium Chloride:
\[ [Na^+] = 154 \times 2 \text{L} = 308 \]
\[ [Cl^-] = 154 \times 2 \text{L} = 308 \]
\[ \text{Dilutional } [\text{SID}] = 30 + 2 \text{L} = 37.0 \text{ (more alkalosis)} \]

TBW 50 kg.
TBW 60% = 0.6 \times 50 \text{ kg} = 30 \text{L}
\[ [Na^+] = 140 \times 30 \text{.140} = 4200 \]
\[ [Cl^-] = 100 \times 30 \text{.100} = 3000 \]

Normal plasma
\[ [\text{SID}] = 40 \]

Rapid Saline Infusion Produces Hyperchloremic Acidosis

1. Saline produce more acidosis than in LR group
2. BE more negative in Saline group
3. [SID] fall because Saline produce Increase in [Cl^-] more than [Na]
4. [SID] fall because Saline produce Increase in [Cl^-] more than [Na]

DESIGNING ‘BALANCED’ CRYSTALLOIDS

• Large volumes of intravenous saline tend to cause a metabolic acidosis
• To counteract this side effect, a number of commercial crystalloids have been designed to be more ‘physiologic’ or ‘balanced’
• They contain stable organic anions such as lactate, gluconate, malate and acetate (metabolizable anion)

BALANCED CRYSTALLOID

Balanced crystalloid is a solution who have zero [SID] before infusion and have an effective [SID] after the metabolizable anion was metabolized
DESIGNING ‘BALANCED’ CRYSTALLOIDS

- Balanced crystalloids thus must have a [SID] lower than plasma [SID] but higher than zero (about 24mEq/l) to counteract the progressive $A_{\text{TOT}}$ dilutional alkalosis during rapid infusion.
- In other words, Saline can be ‘balanced’ by replacing 24mEq/l of Cl$^-$ with various organic metabolizable anions such as Lactate, Malate, Acetate, Gluconate and Citrate as weak ion surrogates.
- These metabolizable anions underwent rapid metabolized in the plasma after infusion resulting only small increase the plasma Cl$^-$ and then small change in plasma [SID].

THE [SID] OF BALANCED CRYSTALLOID, PLASMA AND SALINE

![Table of [SID] concentrations in different fluids: Ringer Lactate, Ringer Acetate, Ringer Fundin, Plasma, Saline.](image-url)

- Strong Cations
- Strong Anions
- [SID] replaced by metabolizable Anions except in saline

George 2015
DESIGNING ‘BALANCED’ CRYSTALLOIDS

- The principles laid down by the late Peter Stewart have transformed our ability to understand and predict the acid–base effects of fluids for infusion
- Designing fluids for specific acid–base outcomes is now much more a science than an art

**Simple analogy**

**How does bicarbonate increase the pH?**

![Diagram showing the effect of bicarbonate on pH]

The [SID]↑: from 10 to 35: → Alkalosis, pH back to normal → but the mechanism not because we give the bicarbonate but we give Sodium without strong anion like Chloride, so the [SID]↑ → alkalosis
BODY pH REGULATION: Interaction Between Membranes

SERIES OF EVENT OF ELECTROLYTE AND ACID-BASE REGULATION IN THE GI TRACT

• The GI tract is important in acid-base balance because it deals directly with strong ions. It does so differently in different regions along its length, so it’s useful to consider four separate parts that are quantitatively important in their effects on plasma [SID]

• There are four important parts (region);
  • Stomach (Event 1)
  • Pancreas (Event 2)
  • Duodenum (small intestine) (event 3)
  • Colon (large intestine) (event 4)
1. Physiologically, Cl⁻ is secreted into the lumen as a gastric acid. It leaves the plasma temporarily and will return to plasma when it absorbed in the small intestine.

Notes: [SID] of gastric acid become very negative (acidosis)

EVENT 1

3. The consequences is the plasma [SID] at the gastric site will increase → alkalosis

4. In case of prolong vomiting, Cl⁻ will leaves the body and it will decrease the plasma [SID] due to hypochloremia (metabolic alkalosis)

5. Fluid therapy using Saline is more appropriate

George, 2003

1. Cl⁻ will continue passing to duodenum

2. Bile and pancreatic secretion contain large amount of sodium (cations) to neutralize the Cl⁻ in duodenum to prevent the acidifying process

EVENT 2 & 3

3. The [SID] of the intestine fluids become normal

4. Cl⁻ return to plasma site when it reabsorbed in jejunum

5. Plasma [SID] at the intestine site become acidosis because cations and sodium from pancreas will absorb in the colon

George, 2003
EVENT 4
Diarrhea

---

Notes. During diarrhea, intestinal fluids pass through the colon too fast to be properly processed, therefore water and cations have lost from the body → metabolic acidosis.

Notes. Balanced fluids or Lactate Ringer is more appropriate for fluid therapy in metabolic acidosis during diarrhea.

1. Cations and Na return to plasma together with water absorption in the large intestine (colon).


George, 2003
STRONG ION DIFFERENCE IN KIDNEY

THE KIDNEYS ARE THE MOST IMPORTANT REGULATOR OF [SID] FOR ACID-BASE PURPOSE

George, 2015
# EFFECT OF DIURETICS IN URINE COMPOSITION

<table>
<thead>
<tr>
<th></th>
<th>Volume (ml/min)</th>
<th>pH</th>
<th>Sodium (mEq/l)</th>
<th>Potassium (mEq/l)</th>
<th>Chloride (mEq/l)</th>
<th>SID (mEq/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No drug</td>
<td>1</td>
<td>6.4</td>
<td>50</td>
<td>15</td>
<td>60</td>
<td>1</td>
</tr>
<tr>
<td>Thiazide diuretics</td>
<td>13</td>
<td>7.4</td>
<td>150</td>
<td>25</td>
<td>150</td>
<td>25</td>
</tr>
<tr>
<td>Loop diuretics</td>
<td>8</td>
<td>6.0</td>
<td>140</td>
<td>25</td>
<td>155</td>
<td>1</td>
</tr>
<tr>
<td>Osmotic diuretics</td>
<td>10</td>
<td>6.5</td>
<td>90</td>
<td>15</td>
<td>110</td>
<td>4</td>
</tr>
<tr>
<td>Potassium-sparing diurtics</td>
<td>3</td>
<td>7.2</td>
<td>130</td>
<td>10</td>
<td>120</td>
<td>15</td>
</tr>
<tr>
<td>Carbonic anhydrase inhibitors</td>
<td>3</td>
<td>8.2</td>
<td>70</td>
<td>60</td>
<td>15</td>
<td>120</td>
</tr>
</tbody>
</table>

Loop Diuretics (Furosemide) increase the excretion of Cl⁻ via urine → reducing urine [SID] and increasing the plasma [SID] → alkalosis

Renal Compensation for Chronic Respiratory Acidosis

1. Increase CO₂ → increase the [H⁺]
2. ↑NH₄Cl in urine
3. Hypochloremia
4. Hypochloremia increase [SID] → decrease [H⁺]
RENAL COMPENSATION FOR CHRONIC RESPIRATORY ACIDOSIS

- In stable COPD patients, the plasma pH is preserved closer to normal values in blood through a secondary metabolic compensation by increasing the [SID]. Changes are mainly caused by decreasing plasma Cl⁻ or hypochloremia.

RENAL & RESPIRATORY COMPENSATION FOR NON RENAL METABOLIC ACIDOSIS (UA) IN STEWART’S TERM


Non Renal
Met Acidosis (UA); Shock, MODS
Plasma UA → decrease the [SID] → increase the [H⁺]

H⁺ [SID] HCO₃⁻
Na⁺ 140 Cl⁻ 100
pH ↓ 1. Early compensation

Brain Stem
Removal CO₂
hyperventilation

Removal Chlor⁻

Hypochloremia

H⁺ [SID] HCO₃⁻
Na⁺ 140 Cl⁻ 100
pH ↓ 2. Late compensation

NH₃ Sintesis ↑
(Ammoniagenesis)

Liver
Kidney

Hyperventilation → decrease [H⁺]

Hypocloremia will increase [SID] → decrease [H⁺]

George 2015
SEVERE HYPERLACTATEMIA IS MASKED BY ALKALINIZING PROCESSES (HYPOCHLOREMIA) THAT NORMALIZE THE [BE]

Predominant COPD

Predominant shock

Normal [BE] 34 (20%)

Low [BE] 134 (80%)

CONCLUSION

- \([H^+]\) in the plasma is determined by \([SID]\), \(PCO_2\) and \([Atot]\) in the plasma.
- The strong ion composition of the diet, the function of the GI tract and the function of other tissues may alter plasma \([SID]\) from its normal value.
- Plasma \([SID]\) changes by plasma interaction with interstitial fluid through tissue capillary membranes. Interstitial fluid in turn may interact with intracellular fluid through cell membranes.
- Respiration in the lungs and general body circulation regulate alveolar and circulating plasma \(PCO_2\).
- The kidney regulate circulating plasma \([SID]\) by differential reabsorption of \(Na^+\) and \(Cl^-\).
- When circulating plasma \([H^+]\) changes due to \(PCO_2\) changes, the kidneys slowly produce compensating \([SID]\) changes.
- When circulating plasma \([H^+]\) changes due to \([SID]\) changes, respiration in the lungs changes so as to produce compensating plasma \(PCO_2\) changes.
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THINKING ABOUT FLUID IN STEWART’S APPROACH

EASY WAY TO UNDERSTAND STEWART’S ACID-BASE