TUTORIAL ON ACID BASE STEWART APPROACH
CHAIERD WORKSHOP WITH Dr ROOP KISCHEN

1.- Previous Ways of Assessing Acid-Base

2.- Stewart-Fencl: Principles

3.- Chemical Background of Stewart-Fencl

4.- Why Should we Using Stewart-Fencl?

5.- How to Calculate the SIG at The Bedside?

6.- Conclusions-Perspectives

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Ways to assess acid base disorders

- Arterial Blood Gases
- Boston Approach
- Copenhagen/Danish Approach
- Anion Gap Approach
- Stewart approach

Henderson IJ. 1908;21:427-428
Hasselbalch KA. Biochem 1916;78:112-144
Boston Approach

- Base on pioneering work of Henderson and Hasselbalch
- Henderson (1909) - applied law of mass action to carbonic acid reaction
- $\text{H}^+ \text{ relationship with } \text{CO}_2 \text{ and } \text{HCO}_3^-$
- Sörensen (1909) introduced pH logarithmic scale
- Hasselbalch (1916) modified Henderson’s equation

Severinghaus JW et al. JAP 1956;9:197-200
Boston Approach

- Tufts University, Boston
- Based on H H equation
- Developed after studying patients with known but stable conditions
- “Allows to classify disorders” according to primary acid type
- Respiratory VS metabolic
- Calculation of resting PaCO$_2$ in chronic respiratory disorders
- Unmeasured anions = drop in bicarbonate
- Cannot tell extent or aetiology of disturbance
- Used by physicians; Not recommended in the critically ill

Refinements – 1-Buffer Base

- $\text{HCO}_3^-$ changes with $\text{PaCO}_2$
- Many do not recognise this!
- $\text{HCO}_3^- \neq$ determine metabolic acidosis
- Empirically derived equation devised
- Singer & Hastings (1948) - buffer base
- Buffer base = sum of all plasma buffer ions i.e., $\text{HCO}_3^-$ & non-volatile buffers

Singer RB, Hasting AB. Medicine 1948; 27: 223-242
Danish Approach

- Danish experiences with Polio epidemic
- Mortality ≈ 90% in patients with respiratory failure
- Strange ‘metabolic alkalosis’
- Kidneys inability to excrete bicarbonate!
- Later shown to be ↑ HCO$_3^-$ because of CO$_2$ retention
- Suddenly physicians are now awake seeking better ways of assessing acid-base balance
Refinements – 2 & 3-Base Excess

- Astrup (1960) – standard bicarbonate
- $\text{HCO}_3^-$ at $\text{PaCO}_2$ - 40 mmHg (5.33 kPa)
- Siggard-Andersen & Engle (1960)
- Base Excess (BE)
- $\Delta \text{Hb}$ affect BE
- \textit{In vitro} VS \textit{In vivo} values
- Siggard-Andersen (1977) – ‘Van-Slyke’ equation

Danish Approach

- All is not well
- BE, although stable, changes with \( \text{PaCO}_2 \)
- Standard BE
- Haemoglobin changes effect this as well
- Refined by assuming Hb value of 5 g/dl
- No accounting for changes in \( A_{\text{TOT}} \)
- Attempted correction applied
Anion Gap Creation & Limitations

- AG = ([Na⁺] + [K⁺]) – ([Cl⁻] + [HCO₃⁻])
- Usually positive; unmeasured anions (12-16 mmol/l)
- For unmeasured anions (lactate, PO₄, ketones)
- ↑ AG = usual in lactic acidosis, ketosis, poisonings
- No ↑ in AG = e.g., HCMA
- May be of value in stable patients
- Artificial value; Not a physiological reality
- Low albumin affects the value
- Of limited value in the critically ill

Stewart (Modern) Approach

- Not really modern
- Stewart proposed it in 1978
- Modified by Figge, Fencel, Watson, Constable & Kellum
- Based on principles of physical chemistry
- Applied to complex body fluids
- Water, Solutions of CO$_2$, Weak acids, strong ions dissolved in water

Stewart PA. Respi Physiol 1978;33:9-26
The Stewart Approach: Principles

- HCO3- is NOT the CAUSE of the Problematic.

- HCO3- Variations are well only Consequences.

- pH rely on independent Factors
  - SID
  - Weak Acid Total Concentration (Atot)
  - PCO2

Simplified Stewart Fencl Approach: Background

Potential Advantages:

- Classical Approach has several Drawbacks:
  - Do not take account of non bicarbonate Buffers
  - Not mechanistic
  - Do not quantify acidosis
  - Too limited in complex situations. Acidosis without lactate

RULES:

- Water is an important source of H+
- Ionic Strength: Strong and Weak Ions
- Electrical Neutrality-Electrochemical Dissociation
- Dependent & Independent variables
Simplified Stewart Fencl Approach: Why?

Equilibrium formula:

\[ H_2O + CO_2 \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^- \]

...you’ll notice that HCO_3 can be affected by respiratory (CO2) or metabolic (H+) components, and therefore isn’t a specific marker for either.

In fact, the relationship between metabolic acidosis and bicarbonate is **neither consistent nor linear**.

Simplified Stewart Fencl Approach

You’ll read everywhere that pH is “tightly controlled” by the body in a “narrow range.”

Logarithms introduce a false sense of tight clustering. When the pH changes by 0.3 units, (say from 7.5 to 7.2), this represents a doubling of the hydrogen ion concentration (from 40 nMol/L to 80). Even normal variation between 7.35 and 7.45 represents 25% variation in \([H^+]\). That is not “tight.”

Fencl V et al. AJRCCM 2000;162:2246-2251
The difference between the strong cations and strong anions is called the Strong Ion Difference (SID), and indicates the net ionic charge of the weak anions; so it indicates the relative strength of $H^+$ and $HCO_3^-$. 

Fencl V et al. AJRCCM 2000;162:2246-2251
Plasma Electro-Neutrality

Cations:
- Na+
- K+
- Ca++
- Mg++

Anions:
- Cl-
- HCO3-
- UA-
- Alb-
- Pi-

SID
Calculate sodium-chloride effect:

- **SBE NaCl** = ([Na+] – [Cl-]) – 38

  Where 38 is the average normal strong ion difference i.e., difference between normal values of sodium and chloride; K+, Mg++ and Ca++ being ignored for simplification.

Where: **SBE** = Standard Base Excess (that we normally obtain from blood gas machines)

- **SBE Calc** = SBE NaCl + SBE Alb

- **Unmeasured anion effect or SBEG** = SBE – SBECalc

Figge J, Fenclo V. J Lab Clin Med 1992;120:713-719
- Calculate albumin effect, \( SBE\ Alb = 0.25 \times (42 - \text{measured albumin in blood}) \) \{Where 42 is normal plasma albumin\}.

- \( SBE\ Calc = SBE\ NaCl + SBE\ Alb \)

- Where: \( SBE = \text{Standard Base Excess} \) (that we normally obtain from blood gas machines)

- Unmeasured anion effect or \( SBEG = SBE - SBE\ Calc \)

Figge J, Fencl V. J Lab Clin Med 1992;120:713-719
Bedside calculations:

- **NaCl effect** = \([Na^+] - [Cl^-]\) – 38 \(^{(1)}\)
  
  {where 38 is normal average difference in strong ions – Na and Cl}

- **Albumin effect** = 0.25x \([42 – \text{albumin}]\) \(^{(2)}\)
  
  where 42 is normal serum albumin

- Thus true BE = BE – \([1 + 2]\)

- Works by the bedside!

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Saline Infusion: Effects on SID & SBE

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Plasma Electro-Neutrality

**Cations**
- Na+
- K+
- Ca++
- Mg++

**Anions**
- Cl-
- HCO3-
- Alb-
- UA-
- Pi-

**SID**
Towards Acidosis

- SID
- PaCO₂: 40 torr
- Album: 40 g/l
- Phosph: 0.8 mmol/l
- HCO₃⁻: 17 mEq/l
- pH: 7.25

Towards Alcalosis

- SID
- PaCO₂: 40 torr
- Album: 40 g/l
- Phosph: 0.8 mmol/l
- HCO₃⁻: 29 mEq/l

Lactic Acidosis

Levrault, Payen
Intensive Corpus on Acid Base, France 2011
**Hypoalbuminemia**

- **PaCO$_2$**: 40 torr
- **Album**: 10 g/l
- **Phosph**: 0.8 mmol/l
- **HCO$_3^-$**: 33 mEq/l
- **pH**: 7.54
- **SID**: 39 mEq/l
- **Alb**: 4.2 mEq/l
- **Phosph**: 2.2 mEq/l

Towards Acidosis

Towards Alcalosis

Levrault, Payen
Intensive Corpus on Acid Base, France 2011
Towards Acidosis

- PaCO₂
- Album
- Phosph
- pH 7.40
- HCO₃⁻ 24 mEq/l

Towards Alcalosis

- SID
- 40 torr
- 10 g/l
- 0.8 mmol/l
- 29 mEq/l

**Hypoalbuminemia & Lactic Acidosis**

Levrault, Payen
Intensive Corpus
on Acid Base, France 2011

Alb⁻ 3.5 mEq/l
Phosph⁻ 1.8 mEq/l
Conclusions & Perspectives

- SBE versus SIG-Bicarbonate is not anymore a player but a consequence.
- Small Changes in pH are in fact huge changes in [H+] and Stewart–Fencl can much better evaluate this.
- In Hasselback, pH is not specific of metabolic problems and a metabolic can be easily blunted.
- Due to the principle of electroneutrality, one can easily understand why hyperchloremia can induce acidosis.
- Albumin and Cl- are extremely important players in the Stewart-Fencl Approach.
- Acidosis without lactate can be detect easier with Fencl.
- The use nowadays of citrate can benefit a lot of this approach especially in case of citrate intoxication.