Inter Pretation of Acid Base Disturbance in Critically ill Patients.

By :-

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M.D.

- Normal Blood PH

  7.35 to 7.45
- Crucial importance to maintain homeostatic function of Body.
- Any Significant change relates to change in H + ion con. in Plasma
- It Ph <7 or >7.8 → Imminent

  danger

  ↓

  Death

Concept of Ph

- Acidity of Aqueous solution measured by H+ ion concentration.
- Notation of Ph is negative logarithm of H + ion Concentration
- More H +ion → less Ph

Less H +ion → high Ph

- H + ion concentration in extra cellular fluid determined by Balance between Pco2 and Hco3 concentration in the fluid

Follow equation

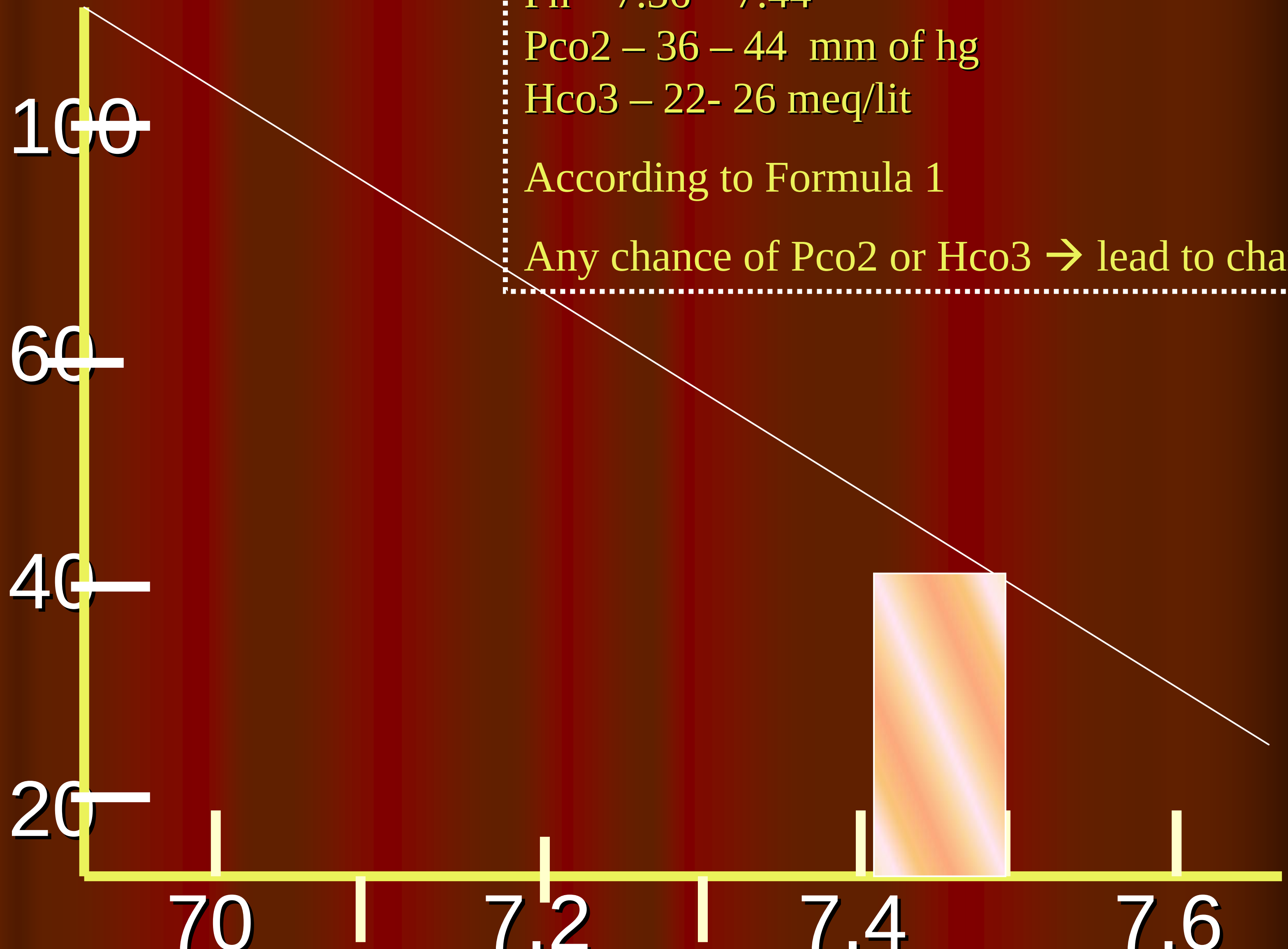
H+ = 24 x Pcox / HCo3
(neg/L)

Using normal arterial Pco2=40 mm of hg
Hco3=24 Eq/L

H += 24x40 = 40 neg/Lit
24

H+ = 40neg/L → Correlates with PH 7.4

→ H +ion and Ph are inversely related.



Normal range

Ph – 7.36 – 7.44

Pco2 – 36 – 44 mm of hg

Hco3 – 22- 26 meq/lit

According to Formula 1

Any chance of Pco2 or Hco3 → lead to chance in PH

- ➲If change involves Pco2 → Respiratory acid base disorder.
- ⇧Pco2 → Respiratory Acidosis
- ⇧Pco2 → Respiratory Alkalosis.
- ➲If Chance involves Hco3 → Metabolic

Acid Base disorder.

- ⇩Hco3 → Metabolic Acidosis
- ⇧Hco3 → Metabolic Alkalosis.

PH ↓ 7.36 → Acidemia

PH ↑ 7.44 → Alkalemia

- Tignt Control of Ph → Fairly constant values of Pco2 & Hco3.
- Change in any one → Proportionate change in other to keep Ph constant.
- Inc Pco2 → inc Hco3 → PH Constant.
- Respiratory disorders (change in Pco2)

  initiates

  Changes in HCo3- Metabolic response
- Initial Chance in Pco2 or Hco3 is Primary

  Acid Base disorder.
- Subsequent response → Compensated or Secondary Acid Base disorder.

Primary Disorder	Primary Chance	Compensatory Chance
Respiratory Acidosis	↑ PCo2	↑ HCo3
Respiratory Alkalosis	↓ PCo2	↓ HCo3
Metabolic Acidosis	↓ HCo3	↓ PCo2
Metabolic Alkalosis	↑ HCo3	↑ PCo2

- Metabolic acid Base disorder → Prompt Ventilatory response

Peripheral chemoreceptor (Carotid today)

- Metabolic acidosis → Stimulates Chemoreceptor

↓

Ses Ventilation

↓

Ses PCo2

- Metabolic Alkalosis → Silences Chemoreceptor

↓

↓ Ses Ventilation

↑ ↓ PCo2

Useful Formulas for Acid Base interpretation

(1) Metabolic acidosis → Expected PCo2 = (1.5xHco3) + 8+2

(2) Metabolic Alkalosis → Expected Pco2= 0.7xHco3+21+2

(3) Acute respiratory → Expected PH= 7.4=(.008 x)
acidosis

(4) Acute respiratory alkalosis→ Expected PH = 8.4 + (0.008x(40-Pco2)

(5) Chronic respiratory → Expected PH = 7.4 (.003xPco2-40) acidosis

(6) Chronic respiratory → Expected PH = 7.4 + (0.003x40-Pco2)
Alkalosis

Compensation for Metabolic Acidosis

Metabolic acidosis

↓

Tachypnoea

↓

↓ CO_2

↓ CO_2 → may be Compensated or Uncompensated

In a Patient of Metabolic acidosis

S. HCo3 is 15meq/L.

Expected Pco2 = (1.5xHco3) + 8+2

= (1.5 x 15) + 8 +2

= 30.5 +2

If Pco2 is same then it is Compensated metabolic acidosis

If Pco2 is >30.5 + 2 than there is Superimposed respiratory acidosis

If Pco2<28.5 than there is associated respiratory alkalosis.

Compensation for Metabolic Alkalosis

Expected Pco2 = 0.7x Hco3+21 +2

In a patient of Metabolic Alkalosis with

Hco3 = 4omeq/L

Expected Pco2 = 0.7x 40 + 21+2

Pco2 = 28 + 21+2

= 49 +2 mm of hg

If measured Pco2 is equal to expected Pco2 than – Compensated metabolic Alkalosis

If Pco2 is ↑ than Metabolic alkalosis + respiratory acidosis

If Pco2 ↓ than Metabolic Alkalosis + respiratory alkalosis.

- Compensatory changes of Hco3 in response to Pco2

↓ Occur in kidneys

↓ takes 6 to 12 hrs to develop

Due to this

Respiratory acid Base disorder → Acute / Chronic

Acute Before Compensation

Chronic After Compensation

✍ Expected arterial Ph in Acute cases

Patient → PH – 7.4

Pco2 increases from 40 to 60

Arterial PH = 7.4 = (0.008 x Pco2 – 40)

= 7.40 – 0.16

= 7.24

If Pco2 decrease from 40 to 20

Then arterial PH = 7.4+ 0.008 x 40 – Pco2

PH = 7.40 + 0.16

PH = 7.56

✍ <u>Expected arterial Ph in Chronic cases</u>

Patient of emphysema + Co2 retention Pco2 60 mm of hg

Expected Ph = 7.4 – 0.003 x Pco2 - 40

= 7.4 – 0.006

= 7.34

In acute rise → expected Ph was 7.24 due to renal Compensation Ph in chronic case is 7.34.

- Rule 1 Acid Base abnormality is present if either PaCo2 or PH is outside normal range.
- Rule 2 If PH &Pco2 are both abnormal see directional change.
  - If both increase or decrease in same direction – disorder is metabolic
  - If both change in opposite direction disorder is respiratory

- PH – 7.23

Pa Co2 – 23 mm of hg

PH & Pa Co2 are both ↓

So Pt has metabolic acidosis

Rule 3

If PH or PaCo2 is normal

- Mixed metabolic and respiratory acid Base disorder.

If PH is normal – direction of change of PaCo2

↙ Identifies respiratory acidosis of alkalosis

If paCo2 in normal → direction of change in PH

↙ identifies metabolic disorder

In Primary metabolic acidosis

Use measured HCo3 → to Calculate expected Pco2.

- If measured Pco2 = expected Pco2 – fully compensated
- If measured Pco2>expected Pco2 – Superimposed respiratory acidosis
- If measured Pco2 < expected Pco2 – Superimposed respiratory alkalosis.

- PH – 7.32

  PCo2 – 23 mm of hg

  HCo3 – 15 meq/L

PH. Hco3   Metabolic acidosis

Expected PCo2 = 1.5x15 + 8+2

= 22.5 + 8 +2

= 30.5 +2

Measured PCo2 is 23 < Expected PCo2 is 30.5

- Superimposed respiratory Alkalosis on metabolic acidosis.

- In Respiratory acidosis or Alkalosis use Paco2 to calculate expected PH.
- Compare measured PH to expected PH
- Decide if Condition is
  - Acute
  - Partially Compensated
  - Fully Compensated
- **In respiratory acidosis**

If measured PH < expected PH then there is Superimposed metabolic acidosis.

- If measured PH> expected Ph then Superimposed metabolic alkalosis.

In respiratory alkalosis

If measured PH<expected Ph then Superimposed metabolic acidosis

If measured PH>expected PH then superimposed metabolic alkalosis.

PH 7.54

PCo2 23

PaCo2 & PH are chanced in opposite direction.

So Problem is respiratory alkalosis.

Expected Ph = 7.4 + 0.008 x (40-23)

= 7.54

- Condition is Uncompensated respiratory Alkalosis.

Anion Gap → Estimate of relative abundance of Unmeasured anions.

Used to determine if lactic acidosis

Due to accumulation of non volatile acids

Loss of Bicarbonates (Diarrhoea)

Anion gap → (UA – UC) = Na - (CL+HCo3)

UA= Unmeasured Anions

Uc = Unmeasured cations

Normal AG= 12+4 meq/L

Influence of Albumin

- Albumin → major source of unmeasured anions.
- 50% ↓ Albumin → 75% reduction in anion gap.
- Hypo Albumenia → Common in ICU
- Influence of Albumin on AG is important.

Adjusted AG = observed AG+2.5x(4.5- measured Albumin G/DL)

Pt → Calculated AG→ 10 meq/L

Serum Albumin 2 g/dl

Adjusted AG = 10 +(2.5 x 2.5) = 16 meq/L

60 % increase

- Seemingly normal AG → transformed in elevated AG.
- Important formula in Pts of ICU ↓Albumin.

Common causes of Metabolic acidosis

High AG acidosis	Normal AG acidosis
Lactic acidosis	Diarrhoea
Keto acidosis	Isotonic saline infusion
End stage Renal failure	Early renal insufficiency
Methanol injestion	Renal acidosis
Ethylene glycol ingestion	Acetazolamide
Salicylate toxicity	Ureteroenterostomy.

High Amiongap

Fixed acid → added to intracellular space

H+ions Anions

H+ combines HCo3 → Carbonic acid

HCo3 → leads to high Anion Gap

- In Metabolic acidosis → loss of Bicarbonate from extra cellular fluid
- Countered by gain of Chloride ions to maintain electrical charge neutrality
- Chloride – Proportional → loss of Bicarb
- So AG = Na – (CL + Hco3) remain constant.
- CL increase → termed hyper chloremic metabolic acidosis.

C/f → Metabolic acidosis

- →Hyperventilation (stimulation of Res Centre)
- →Obtunded Mental State (Drowsiness → deepcoma)
- →Smell of ketones or Ammonia
- →Progressive deterioration of clinical state

Lactic acid acidosis

Lactic acid → Product of Glucose metabolism.

Most common causes

- Sepsis
- Septic shock
- Cardiogenic shock
- Hypoxia
- Severe Anemia
- Severe Liver cell dysfunction
- Thiamine deficiency.
- D Lactic acid Acidosis (Dextro isomer) bacillus fragilis,gm-ve
- Severe respiratory or metabolic Alkalosis
- Drugs → epinephrine
- → Nitro pruside

Diagnosis –

- ABG
- High Anion Gap >30
- If PH < 7.25

Urgent Mx required.

In any acidosis

- Treatment → for the Basic cause
- Important to temporarily current acidosis by giving 7% Soda BicarbSoln.
- Improves Pump function
- Improves Systemic Blood Pressure
- Improved Perfusion to tissues.

Sodium Bicarb replacement

Formula → Body wt x 0.3 x (desired HCo3- serum Hco3)

- Give 50% as initial bolus
- Rest replace as infusion in 4 to 6 hrs.
- In associated respiratory acidosis

  correct respiratory acidosis

  then tackle metabolic acidosis.

Metabolic Alkalosis

Aeti

→ Vomiting or Nasogastric suction

- ✓ loss of gastic juice(50-100meg/L of H +ion)
- ✓ Also associated loss of Cl, Nat, K+ &H2o

Diuretics (Furosemide)

❖ **Loss of**

↓ Chloride
Water
Nat
K+

Chloride replaced by Bicarb → absorbed → leads to alkalosis

- ❖ ↑ In Crease K+ loss → H+ in distal tubule-- alkalosis
- ❖ Water loss → Aldstone → Promotes loss of k+ &H+

Administration of excess Alkali

orally

I/V

- Renal dysfunction – Prevents Bicarbs excretion
- Associated electrolyte dysfunction (Chloride depletion)
- Over enthusiastic use of 1/v soda Bicarb
- Corticosteroid therapy

Adrenocortical hyperfunction

Aldosteronism

Well marked hypokalemia

Metabolic Alkalosis

- Mental obtundation
- Seizures
- Cardiac dysfunction
- Cardiac arrhythmias
- Ventilation may be depressed (secondary increase of paCo2)
- Shifts o2 curve to left
- ses delivery of o2 to tissues.

↓

Disastarous consequence.

Management

- Almost always → chloride responsive
- Replacement by N saline
- Hypokalemia → KCL
- Mineralocorticoid excess → Aldactone

Formula – Chloride deficit

= 0.27 x wt x (100 – measured)

Volume of saline in Lit = $\frac{\text{Cl deficit}}{154}$

Use of HCL

Rare instances When Alkalosis not corrected

- Saline
- K+ replacement

0.1 N HCL Soln Used

Infusion – 0.2meg/K/hr

- Very dangerous
- Thrombophlebitis
- Continuous arterio venous hemofiltration

THANK YOU