Acid-base disorders

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Introduction

- Body pH is mainetained in the narrow interval (6,8 – 7,8) with the help of extra- and intracellular buffer systems.
- The most important extracellular buffer is bicorbanate (HCO_3^-):

$H^+ + HCO_3^- <-> H_2CO_3 <-> H_20 + CO_2$

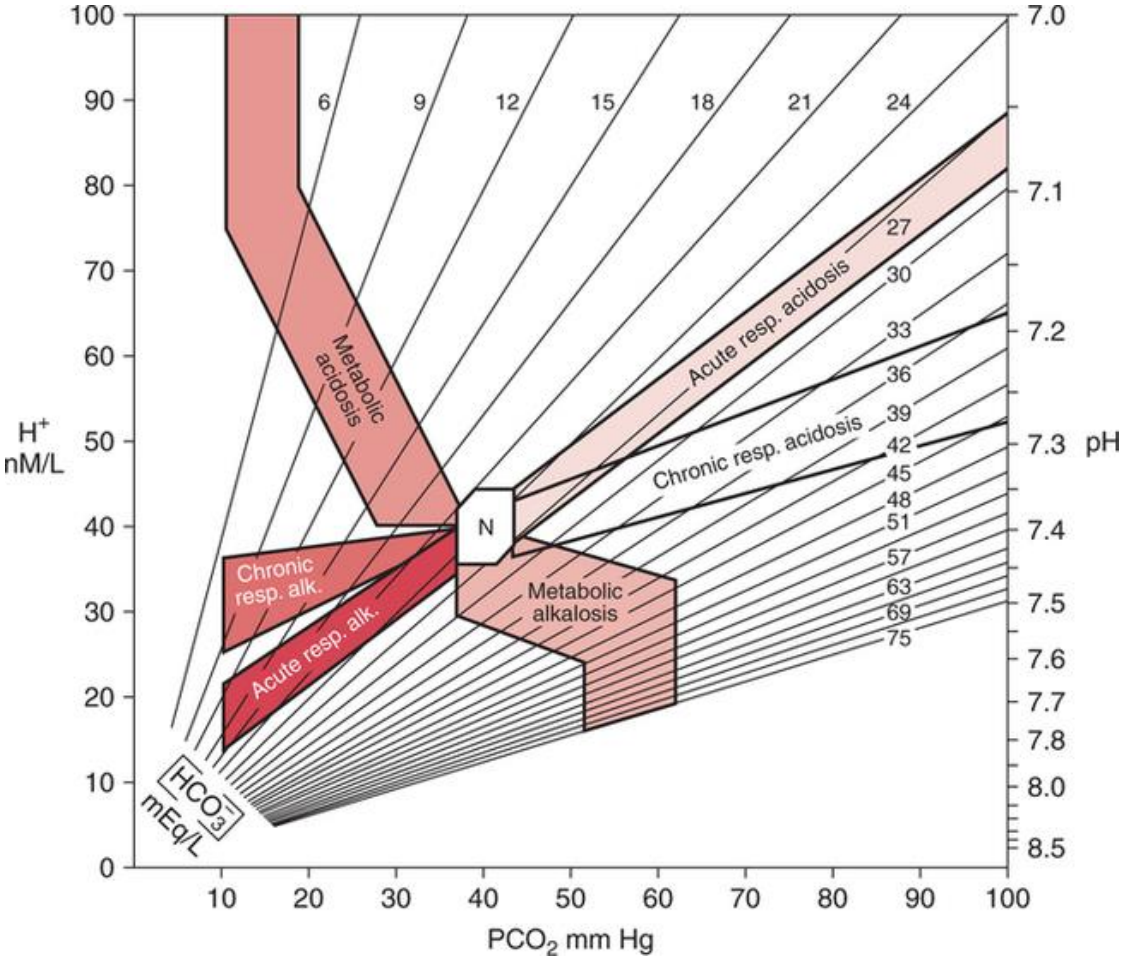
- The relationship maybe expressed:

$[H^+] = K'a \times 0,03 \times P\ co_2 / [HCO3^-]$

- Same equation in logarithmic terms – Hendersons-Hasselbach equation:

$pH=6,1 + \log([HCO3^-] / 0,03 \times P\ co_2)$

Acid-base map



Clinical blood gases 2nd ed., ed. by W. J. Malley, 2005

Case 1: history

- 54 yo male in ER, was found lying on the floor for unknown time
- Anamnesis: alcohol abuse, 3 y after sigma resection due to neoplasia
- Lethargic, pale, reduced skin turgor, RR 24’, TA 88/52 mmHg, HR 112’, oligo-anuric

Case 1: lab

Urea	29,44 mmol/l	pH	7,77
Crea	601,09 mcmol/l	pCO_2	34 mmHg
Glu	7,54 mmol/l	pO_2	56 mmHg
CRP	12,84 mg/l	HCO_3^-	49,4 mmol/l
Alb	31,1 g/l	Na^+	117 mmol/l
		K^+	3,3 mmol/l
		Cl^-	62 mmol/l
		Lac	3,2 mmol/l

Case 1: q1

Which is the primary acid-base disturbance in the patient?

A Metabolic acidosis

B Metabolic alkalosis

C Respiratory acidosis

D Respiratory alkalosis

Terms

- Acidemia – a decrease in the blood pH (H^+ increases)
- Alkalemia – an elevation in the blood pH (H^+ decreases)
- Acidosis – acidosis process which leads to acidemia
- Alkalosis – process which leads to alkalemia
- However, things may differ in patients with mixed acid-base disorders
- Respiratory causes – primary abnormalities in the Pco_2 pathway which is regulated by the respiration
- Metabolic causes – primary abnormalities in the HCO_3^- pathway

General characteristics of the primary acid-base disturbances

Disorder	pH	[H+]	Primary disturbance	Compensatory disturbance
Metabolic acidosis	↓	↑	↓ $[HCO_3^-]$	↓ Pco_2
Metabolic alkalosis	↑	↓	↑ $[HCO_3^-]$	↑ Pco_2
Respiratory acidosis	↓	↑	↑ Pco_2	↑ $[HCO_3^-]$
Respiratory alkalosis	↑	↓	↓ Pco_2	↓ $[HCO_3^-]$

Respiratory **O**ther **M**etabolic **E**quel

Case 1: q2

Which is the secondary acid-base disturbance in the patient?

A Metabolic acidosis

B Chronic respiratory acidosis

C Acute respiratory acidosis

D Chronic respiratory alkalosis

E Acute respiratory alkalosis

Compensation of the primary acid-base disturbancies

Disorder	Primary change	Response
Metabolic acidosis	↓ **$[HCO_3^-]$**	1,2 mmHg decrease in Pco_2 for every 1 mEq/l fall in $[HCO_3^-]$
Metabolic alkalosis	↑ **$[HCO_3^-]$**	0,7 mmHg elevation in Pco_2 for every 1 mEq/l rise un $[HCO_3^-]$
Acute Respiratory acidosis	↑ **Pco_2**	1 mEq/l increases in $[HCO_3^-]$ for every 10 mmHg rise in Pco_2
Chronic Respiratory acidosis	↑ **Pco_2**	3,5 mEq/l increases in $[HCO_3^-]$ for every 10 mmHg rise in Pco_2
Acute Respiratory alkalosis	↓ **Pco_2**	2 mEq/l reduction in $[HCO_3^-]$ for every 10 mmHg fall in Pco_2
Chronic Respiratory alkalosis	↓ **Pco_2**	4 mEq/l decrease in $[HCO_3^-]$ for every 10 mmHg reduction in Pco_2

Case 1: a2

ABG: 34/49,4/7,77

pCO_2 predicted = 40 + (HCO_3^- - 24) x 0,7 =

= 57,8 mmHg

pCO_2 difference = 57,8 – 34 = 23,8 mmHg

Case 1: q3

What is the main pathopysiological path of primary metabolic alkalosis in the patient?

A Loss of hydrogen

B Retention of bicarbonate

C Contraction alkalosis

Metabolic alkolosis: causes

Loss of hydrogen:

- Gastrointestinal loss (vomiting, antacid therapy, chloride-losing diarrhea);
- Renal loss (loop or thiazide diuretics, hyperaldosteronism, postchronic hypercapnia, low chloride intake, penicillin derivative, hypercalcemia);
- H^+ movment into cells (hypokalemia, refeeding syndrome).

Retention of bicarbonate:

- Massive blood transfusion;
- Administration of $NaHCO_3$;
- Milk-alkali syndrome.

Contraction alkalosis:

- Loop or thiazide diuretics;
- Gastric losses in patients with achlorhydria;
- Sweat losses in cystic fibrosis.

Clinical physiology of acid-base and electrolyte disorders 5th ed., ed. by B. D. Rose, T. W. Post, 2001

Case 1: q4

What is the main pathopysiological path of secondary respiratory alkalosis in the patient?

A Hypoxia

B Pulmonary disease

C Direct stimulation of the medullary respiratory centre

D Mechanical ventilation

Respiratory alkalosis: causes

- Hypoxia (lung diseases, CHF, hypotension, severe anemia, high-altitude resistance);
- Direct stimulation of the medullary respiratory centre (psychogenic hyperventilation, hepatic failure, gram-negative septicemia, salicylate ingestion, neurologic disorders, pregnancy);
- Mechanical ventilation.

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Case 1: q5

How could we predict fluid responsivness in this patient?

A Fluid resuscitation trial

B Measurment of the hydration state with BIA

C Patients with metabolic alkalosis are fluid non-responsive

D By measuring electrolyte level in urine

Urine Cl^- concentration in patients with metabolic alkalosis

Less than 25 mEq/l

- Vomiting and nasogastric suction
- Diuretics (late)
- Factitious diarrhea
- Posthypercapnia
- Cystic fibrosis
- Low chloride intake

Greater than 40 mEq/l

- Mineralcorticosteroid excess
- Diuretics (early)
- Alkali load
- Barterr's or Gitelman's syndrome
- Severe hypokalemia (< 2 mEq/l)

Case 2: history

- 39 yo male in ER, was found unconscious lying on the street
- GCS 10 (E3V2M5), alcohol smell, RR 28’, HR 103’, TA 110/65 mmHg

Case 2: lab

Urea	4,3 mmol/l	pH	7,083
Crea	104,56 mcmol/l	pCO_2	17 mmHg
Glu	10,9 mmol/l	pO_2	89 mmHg
CRP	1,53 mg/l	HCO_3^-	5 mmol/l
Alb	34,6 g/l	Na^+	138 mmol/l
EtOH	50 mg/dl	K^+	4,62 mmol/l
P_{Osm}	336 mOsmol/l	Cl^-	98 mmol/l
		Lac	4,3 mmol/l

Case 2: q1

Which is the primary acid-base disturbance in the patient?

A Metabolic acidosis

B Metabolic alkalosis

C Respiratory acidosis

D Respiratory alkalosis

General characteristics of the primary acid-base disturbances

Disorder	pH	[H+]	Primary disturbance	Compensatory disturbance
Metabolic acidosis	↓	↑	↓ $[HCO_3^-]$	↓ Pco_2
Metabolic alkalosis	↑	↓	↑ $[HCO_3^-]$	↑ Pco_2
Respiratory acidosis	↓	↑	↑ Pco_2	↑ $[HCO_3^-]$
Respiratory alkalosis	↑	↓	↓ Pco_2	↓ $[HCO_3^-]$

Respiratory **O**ther **M**etabolic **E**quel

Case 2: q2

What is the main pathopysiological path of primary metabolic acidosis in the patient?

A Inability to excrete the dietary H^+ load

B Increased H^+ load

C Increased HCO_3^- loss

Metabolic acidosis: causes

Inability to excrete the dietary H^+ load:

- Diminished NH_4^+ production: renal failure, hypoaldosteronism (RTA type 4);
- Diminished H^+ secretion: RTA (distal) type 1;

Increased H^+ load:

- Lactic acidosis;
- Ketoacidosis;
- Ingestions (salicylates, methanol, ethylene glycol, paraldehyde etc.);
- Massive rhabdomyolysis;

Inceased HCO_3^- loss:

- Gastrointestinal losses: diarrhea, high excretory load GI fistulas, ureterosigmoidstomy, cholestyramine;
- Renal loss: RTA (proximal) type 2.

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Case 2:q3

What about the anion gap?

A No need to calculate in this case, clinical problem is clear

B Anion gap maybe calculated in case if mixed acid-base disturbace is suspected

C Anion gap should not be calculated in the primary metabolic acidosis

D Anion gap should always be calculated in the primary metabolic acidosis

Anion gap

Anion gap = $[Na^+] – ([Cl^-] + [HCO_3^-]) =$

$= 138 – (98 + 5) = 35$ mmol/l

N value 5-11 (8) mEq/l

Correction to albumin, reduction of anion gap of 2,5 mEq/l for every 10 g/l decline of plasma albumin

Anion gap measurment in patients with metabolic acidosis

High anion gap

- Lactic acidosis: lactate;
- Ketoacidosis: β-hydroxybutirate;
- Renal failure: sulfate, phosphate, urate, hippurate;
- Salicylate: ketones, lactate, salicylate;
- Methanol: formate;
- Ethylene glycol: glycolate, oxalate;
- Massive rhabdomyolysis.

Normal anion gap

- Diarrhea;
- RTA type 1;
- RTA type 2;
- RTA type 4;
- Ammonium chloride;

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Case 2: q4

Are any other calculations needed to proceed with the diagnosis?

A Urine anion gap

B Urine osmolal gap

C Plasma osmolal gap

D All of them

E Non of them

Urine anion gap

Urine anion gap = $([Na^+] + [K^+]) – [Cl^-]$

- N value positive or near 0
- In metabolic acidosis usually exceeds -20 mEq/l (when renal function is intact)

Should not be used in:

- High anion gap metabolic acidosis
- Volume depletion with avid Na^+ retention

Urine osmolal gap

Should be calculated in the case of positive urine anion gap, to disciminate if excretion of unmeasured anions is responsible by estimating ammonium concentration

Calculated urine osmolality (mOsmol/kg) =

$2 \times ([Na^+] + [K^+]) + [UREA] + [Glucose]$

Plasma osmolal gap

Calculated P_{osm} = 2 x [Na^+] + [Glucose] + [UREA] + 1,25 x [Ethanol] =

= 2 x 138 + 10,9 + 4,3 + 1,25 x 10,85 = 304,8 mOsmol/kg

ΔP_{osm} = 336 – 304,8 = 31,2 mOsmol/kg

- High osmolal gap (> 25 mOsmol/kg) suggestive for methanol or ethylenglycol ingestion

Case 2: q5

If there are any secondary acid-base disturbance in this clinical situation?

A Definetaly no

B Probably yes

C High anion gap metabolic acidosis usually combines with respiratory acidosis

D Should be checked

Δ/Δ

Δ anion gap/Δ plasma HCO_3^- concentration

Δ/Δ in high anion gap acidosis usualy is 1-2

Δ/Δ below 1 suggests combined high and normal anion gap acidosis

Δ/Δ above 2 suggests metabolic acidosis combined with metabolic alkalosis

Compensation of the primary acid-base disturbancies

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Metabolic alkalosis	↑ **$[HCO_3^-]$**	0,7 mmHg elevation in Pco_2 for every 1 mEq/l rise un $[HCO_3^-]$
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Chronic Respiratory alkalosis	↓ **Pco_2**	4 mEq/l decrease in $[HCO_3^-]$ for every 10 mmHg reduction in Pco_2

Case 2: a5

ABG: 17/5/7,08

Δ/Δ = 35 – 8/24 – 5 = 27/19 = 1,42

pCO_2 predicted = 40 + (24 - HCO_3^-) x 1,2 =

= 17,2 mmHg

Case 2: q6

Treatment options?

A Fomepizole i/v

B Ethanol i/v

C Continious Renal Replacement therapy

D Hemoperfusion

E Just supportive treatment

F All answers are correct

Case 3: history

- 57 yo female, in ER compalins about cough with purulent sputum, febrility, somnolence and anxiety
- Anamnesis: Asthma on medical treatment with IGC and LABA
- Body temperature 37,8 ^{o}C, RR 32’, TA 146/84 mmHg, HR 110’, difuse bilateral ronchi on exhalation

Case 3: lab

Urea	6,8 mmol/l
Crea	119,46 mcmol/l
Glu	9,3 mmol/l
CRP	66,5 mg/l
Alb	45,6 g/l

pH	7,15
pCO_2	72 mmHg
pO_2	69 mmHg
HCO_3^-	32 mmol/l
Na^+	146 mmol/l
K^+	5,22 mmol/l
Cl^-	110 mmol/l
Lac	1,3 mmol/l

Case 3: q1

Which is the primary acid-base disturbance in the patient?

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B Metabolic alkalosis

C Respiratory acidosis

D Respiratory alkalosis

General characteristics of the primary acid-base disturbances

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Metabolic alkalosis	↑	↓	↑ $[HCO_3^-]$	↑ Pco_2
Respiratory acidosis	↓	↑	↑ Pco_2	↑ $[HCO_3^-]$
Respiratory alkalosis	↑	↓	↓ Pco_2	↓ $[HCO_3^-]$

Respiratory **O**ther **M**etabolic **E**quel

Case 3: q2

What are the main pathophysiological path of acid-base disturbance in this patient?

A Inhibiton of the medullary respiratory centre

B Muscle weakness

C Airway obstruction

D Disordes affecting gas exchange across the pulmonary capillary

E Other

Acute respiratory acidosis: causes

- Inhibiton of the medullary respiratory centre (opiates, anesthetics, sedative, oxygen in chronic hypercapnia, cardiac arrest, central sleep apnea);
- Muscle weakness (myastenia gravis crisis, periodic paralysis, aminoglycosides, Guillain-Barre syndrome, severe hypokalemia);
- Upper airway obstruction (aspiration of foreign body or vomitus, obstructie sleep apnea, laryngospasm);
- Disordes affecting gas exchange across the pulmonary capillary (ARDS, acute cardiogenic pulmonary edema, severe asthma or pneumonia, pneumothorax);

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Chronic respiratory acidosis: causes

- Inhibition of the medullary center (Pichwickian syndrome, metabolic alkalosis);
- Disorders of respiratory muscles and chest wall (spinal cord injury, poliomyelitis, ALS, MS, myxedema, kyphoscoliosis, morbid obesity);
- Disroders affecting gas exchange (COPD, emphysema, morbid obesity)

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Case 3: q3

In current situation respiratory acidosis is?

A Acute respiratory acidosis

B Chronic respiratory acidosis

C Acute on chronic respiratory acidosis

D Something else

Compensation of the primary acid-base disturbancies

Disorder	Primary change	Response
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Chronic Respiratory alkalosis	↓ **Pco_2**	4 mEq/l decrease in $[HCO_3^-]$ for every 10 mmHg reduction in Pco_2

Case 3: a3

ABG: 72/32/7,15

Predicted HCO_3^- (for acute respiratory acidosis) = 24 + (pCO_2 – 40)/10 =

= 24 + (72-40)/10 = 27,2 mmol/l

Predicted HCO3- (for chronic respiratory acidosis) = 24 + (pCO_2 – 40)/10 x 3,5 =

= 24 + (72-40)/10 x 3,5 = 35,2 mmol/l

Case 3: q4

How would you correct acid-base deisiturbance in this patients?

A Mechanical ventilation

B Non-invasive ventilation

C Oxygen therapy

D Intravenous bicarbonate

E Other

Take home message

- ROME concept
- Always chech for compensatory response
- Always calculate anion gap in metabolic acidosis
- Do not forget to assess electrolyte levels
- Anamnesis and physical examination are crucial

Bona diagnosis, bona curatio

Question?