Acid-Base disorders
Cases

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Case 1

The patient is a 35 year -old female with AIDS brought to the emergency room with a fever of 39^{o}C and a three month history of copious diarrhea.

On physical exam the patient is a well-developed, thin female in moderate distress. Vital signs-(supine) blood pressure 100/60 mmHg, pulse 100/min, respirations 18 bpm and she was afebrile. HEENT (head-eyes-ears-nose-throat) exam was normal. Cardiac exam demonstrated an S1 and S2 without S3, S4 or murmur. Lungs were clear to auscultation and percussion. The abdomen was supple and minimally tender to palpation. Bowel sounds were hyperactive. Stool was guiac negative. Extremities were without cyanosis, clubbing or edema. Neurological exam was intact.

Laboratory Data

Chemistry		Normal Values	Arterial Blood Gas
Sodium	136	136-146 mmol/L	pH 7.35 P_{CO2} 27 mmHg P_{O2} 90 mmHg bicarbonate 14 mmol/L
Potassium	3.4	3.5-5.3 mmol/L	
Chloride	112	98-108 mmol/L	
Total CO_2	14	23-27 mmol/L	
BUN	30	7-22 mg/dl	
Creatinine	1.5	0.7-1.5 mg/dl	
Glucose	105	70-110 mg/dl	

Questions

1. What is/are the critical course of events that is going to alter her acid-base status?

2. What Acid base abnormalities would you expect based on this information?

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Copious diarrhea

- Loss of fluid leading to volume depletion
- Loss of bicarbonate
- Loss of K

2. What Acid base abnormalities would you expect based on this information?

Loss of bicarbonate during diarrhea will lead to metabolic acidosis

3. What physical findings would you expect to see from such an acid base disturbance?

4. Review her blood gases. What is the primary acid-base abnormality? How did you decide that?

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Kussmaul respiration with metabolic acidosis.

4. Review her blood gases. What is the primary acid-base abnormality? How did you decide that?

Metabolic acidosis

- pH is low hence acidemic
- Bicarbonate is low hence metabolic

5. Calculate the anion gap in this patient. What is the normal anion gap. What is the gap due to in normals?

6. What is her bicarbonate gap?

7. Obviously this patient has nonanion gap metabolic acidosis What is the relationship between the bicarbonate gap and the anion gap?

5. Calculate the anion gap in this patient. What is the normal anion gap. What is the gap due to in normals?

Sodium-(Bicarbonate+Chloride)(136-126) = 10 mEq/L

6. Is there a compensatory mechanism for metabolic acidosis? How is that brought about? What are the sensors and effectors for metabolic acidosis?

7. What is the predicted compensatory response?

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Hyperventilation

Acidosis is a strong stimulant to the respiratory center in medulla.

PCO_2 is index of alveolar ventilation.

7. What is the predicted compensatory response?

Expected $PCO_2=(1.5 \times HCO_3)+(8\pm2)=29\pm2$ mmHg

8. Is his respiratory compensatory effort appropriate? Can respiratory compensatory effort fully compensate for metabolic acidosis?

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We see an appropriate level of hyperventilation in an attempt to compensate for metabolic acidosis indicating a normal respiratory system.

This type of assessment is necessary to detect mixed defects.

Respiratory compensation for metabolic acidosis is never complete.

9. Is this a simple or mixed disorder? How did you come to that conclusion?

10. What clinical condition(s) is (are) responsible for the acid-base disturbance in this patient?

9. Is this a simple or mixed disorder? How did you come to that conclusion?

This is a simple disorder

10. What clinical condition(s) is (are) responsible for the acid-base disturbance in this patient?

Diarrhea

11. What are the physiologic mechanisms responsible for the generation of this disturbance?

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- The operative mechanism in a normal anion gap metabolic acidosis is the exchange of bicarbonate for chloride.
- In this case the loss of bicarbonate is along the bowel wall in exchange for chloride.
- The kidney is also avidly absorbing NaCI and water to maintain volume homeostasis.

12. What are the other causes for normal anion gap metabolic acidosis?

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- Renal disease
  - Proximal or distal RTA
  - Renal insufficiency (HCO_3 loss)
  - Hypoaldosteronism / K^+ sparing diuretics
- Loss of alkali
  - Diarrhea
  - Ureterosigmoidostomy
- Ingestion's
- Carbonic anhydrase inhibitors

Case 2

The patient is a 28 year-old female who presents with a complaint of muscular weakness and fatigue. History: bulimia. She has lost 30 pounds (1 pound=0.453592 kg) since her last office visit one year ago. She has no other complaints.

Medications- multivitamins
Allergies-none

On physical exam she is a cachectic female appearing fatigued. Blood pressure 100/76 mmHg, pulse 88/min, respirations 16 bpm and she was afebrile. HEENT exam was remarkable for an erythematous pharynx with scattered excoriations. The remainder of the exam was normal except for her marked weight loss.

Laboratory Data

Chemistry .		Normal Values	Arterial Blood Gas	Urine
Sodium	136	136-146 mmol/L	pH 7.48 P_{CO2} 48 mmHg P_{O2} 80 mmHg bicarbonate 36 mmol/L	pH 6.0
Potassium	2.8	3.5-5.3 mmol/L		
Chloride	85	98-108 mmol/L		
Total CO_2	36	23-27 mmol/L		
BUN	20	7-22 mg/dl		
Creatinine	1.0	0.7-1.5 mg/dl		
Glucose	80	70-110 mg/dl		

Questions

1. What is the primary acid-base abnormality? How did you arrive at that conclusion?

2. Calculate the anion gap? When should we calculate anion gap?

Questions

1. What is the primary acid-base abnormality? How did you arrive at that conclusion?

Metabolic alkalosis

2. Calculate the anion gap.

136-(36 + 85) = 15 mEq/L.

3. What is the hydrogen ion concentration?

4. How did he compensate for metabolic alkalosis?

5. What is the predicted compensatory response?

3. What is the hydrogen ion concentration?

$[H^+] = 24 \cdot (PCO_2/HCO_3)$

$[H^+] = 10^{-pH}$

(24)·(48) /(36)= 32 nM/L

4. How did he compensate for metabolic alkalosis?

Hypoventilation and some acid generation to compensate for the alkalosis.

5. What is the predicted compensatory response?

Expected $PCO_2=(0.7 \times HCO_3)+(21\pm2)=46.2\pm2$ mmHg

6. Is this a simple or mixed disorder? How did you come to that conclusion?

7. What are the common causes for metabolic alkalosis?

6. Is this a simple or mixed disorder? How did you come to that conclusion?

Simple

7. What are the common causes for metabolic alkalosis?

- loss of hydrogen ions from the body
  - vomiting
  - gastric suction
- net rate of renal bicarbonate generation is greater than normal
  - volume contraction
  - potassium depletion
  - increased delivery of sodium to distal tubule (Loop diuretics)
  - minerelocorticoid excess
- Rapid correction of ventilation in a patient with chronic CO_2 retention (posthypercapnic alkalosis)

8. If the urine chloride is < 15 mmol/L, what are the diagnostic possibilities?

9. If the urine chloride is > 25 mmol/L, what are the diagnostic possibilities?

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Sodium chloride responsive metabolic alkalosis i.e. emesis, secondary hyperaldosterone states, etc.

9. If the urine chloride is > 25 mmol/L, what are the diagnostic possibilities?

Sodium chloride nonresponsive states i.e. primary hyperaldosteronism.

10. What clinical condition(s) is (are) responsible for the acid-base disturbance in this patient? What additional information would you seek?

11. What are the physiologic mechanisms responsible for the generation of this disturbance?

10. What clinical condition(s) is (are) responsible for the acid-base disturbance in this patient? What additional information would you seek?

Most likely cause of metabolic alkalosis in this patient is emesis as a result of her bulimia.

11. What are the physiologic mechanisms responsible for the generation of this disturbance?

Loss of hydrochloric acid from the stomach and simultaneous generation of bicarbonate which is added to the systemic circulation from the parietal cells in the stomach.

Case 3

A 68-year-old woman had a cardiopulmonary arrest immediately on returning to the ward after a surgical procedure. The following parameters are those of an arterial blood sample taken approximately five minutes after the catastrophe.

pH=6.85, PCO_2=82 mmHg, PO_2=21 mmHg

$\Delta pH = \Delta PCO_2 \times 0.008 \rightarrow pH = 7.064$

Diagnosis: Combined respiratory and metabolic acidosis.

$HCO_3 = 14$ mEq/L

Na = 136 mEq/L
Cl = 108 mEq/L

AG = 14 mEq/L

What next?

Alb = 2.5 g/dL

AGadj = AG + 2.5 × (4.5 – Alb) → **19 mEq/L**

Lac = 7.0 mEq/L

$\Delta AG/\Delta[HCO_3] = 7/10 = 0.7 <$ **1**

This acid-base disturbance is easily recognized: the high PCO_2 indicates a respiratory acidosis and the low blood HCO_3 signifies a metabolic acidosis. The respiratory acidosis is due to respiratory arrest and circulatory collapse (decreased pulmonary ventilation and perfusion), and the metabolic acidosis is a consequence of the associated tissue anoxia, i.e., anaerobic glycolysis, with excessive production of peripheral lactic acid.

Case 4

The following plasma and blood parameter values are those for a 50-year-old woman who presented semi-comatose at the emergency department. She had been receiving insulin therapy for diabetes mellitus, and over the previous four months had been taking digoxin and chlorothiazide tablets for congestive cardiac failure.

pH = 7.41, PO_2 = 88 mmHg, PCO_2 = 32 mmHg, HCO_3 = 19 mEq/L

K^+ = 2.7 mEq/L, Glu = 1584 mg/dl, ketones = +++

Diagnosis: Mixed respiratory alkalosis and metabolic acidosis.

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K^+ = 2.7 mEq/L, Glu = 1584 mg/dl, ketones = +++

Diagnosis: Mixed respiratory alkalosis and metabolic acidosis.

AG = 34 mEq/L !

The diagnostic feature of this patient's biochemistry values is the combination of mild metabolic acidosis (HCO_3 = 19 mEq/L) and a very high anion gap (34 mEq/L). If we assume that the "personal normal" anion gap for this patient was at the upper limit of the reference interval (17 mEq/L), then the increase in her gap is 17 mEq/L. If this increase was due to diabetic-induced ketosis- the obvious explanation-then the approximate decrease in her plasma HCO_3 would be of the same magnitude. Thus, her original concentration of plasma bicarbonate would have been on the order of 36 mEq/L (19 + 17 mEq/L), i.e., metabolic alkalosis. Furthermore, because most patients who present with diabetic ketoacidosis have a plasma [HCO_3] of around 4 to 10 mEq/L, we can reasonably assume that this patient's concentration exceeded 33 mEq/L before she developed diabetic ketoacidosis. Inspection of her clinical history and of the concentration of potassium in her plasma indicates that she presumably had a metabolic alkalosis, from thiazide therapy and hypokalemia, before the diabetes induced ketoacidosis.

- $\Delta AG/\Delta[HCO_3] = 22/5 = 4.4 >$ **1**

- Gap gap ratio during NS solution administration (?)

Case 5

A 70-year-old man, who over the previous five days had had severe persistent vomiting, was admitted to hospital with severe congestive cardiac failure. On admission he was hyperventilating and was very distressed. He was not taking any medication. His blood gas and plasma electrolyte values at admission are shown below:

pH=7.58, PO_2=154 mmHg, PCO_2=21 mmHg, HCO_3=19 mEq/L, Na^+=130 mEq/L, K^+=5.2 mEq/L, Cl^-=79, Cr=3.5 mEq/L

$\Delta pH = \Delta PCO_2 \times 0.008$ → pH=7.552
$\Delta pH = \Delta PCO_2 \times 0.003$ → pH=7.457

Respiratory alkalosis and metabolic alkalosis….

AG=32 mEq/L

…**and metabolic acidosis .**

The high anion gap suggests an underlying metabolic acidosis. This condition could be due in part to the renal failure and possibly to an accumulation of lactate subsequent to poor tissue perfusion, invariably associated with congestive cardiac failure. The increase in this patient's anion gap is 15 mEq/L above the upper reference interval of 17 mEq/L, and we would expect to see a similar quantitative decrease in his plasma HCO_3. Given his measured bicarbonate value of 19 mEq/L, we suggest that, were he not experiencing metabolic acidosis, his plasma HCO_3 would be about 34 mEq/L (19 + 15 mEq/L), i.e., a metabolic alkalosis. The patient had severe vomiting for five days; thus he very probably also had an underlying metabolic alkalosis.

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Case 6

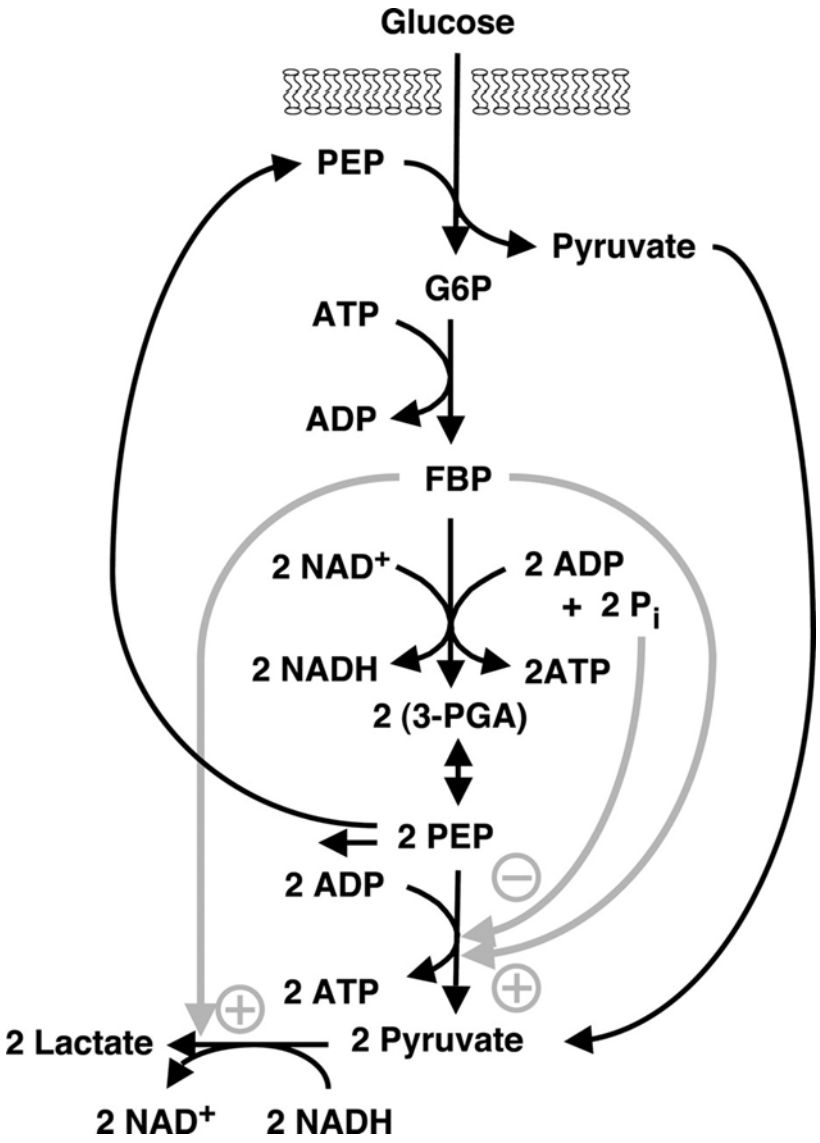
Γυναίκα 28 ετών, με αναφερόμενες διαταραχές συμπεριφοράς,
που δεν λάμβανε ειδική φαρμακευτική αγωγή, βρίσκεται με διαταραχή επιπέδου συνείδησης (GCS: 8/17, E:2, M:5, V:1), χωρίς απόπνοια αιθανόλης.
Αντικειμενικά: ΑΠ 115/60 mmHg, HR=100/min, RR=20 bpm, T=36.5 ^{o}C , SpO_2=99%, CRT<2sec.
Από τον εργαστηριακό έλεγχο βρέθηκαν: Ht=43%, WBC=8500/μl, Ur=36 mg/dl, Glu=108 mg/dl, Na=140 mEq/L, K=4.0 mEq/L, Cl=104 mEq/L, pH=7.29, PCO_2=30 mmHg, HCO_3=14 mEq/L, Alb=4.3 g/dl, Lac=2.1mEq/L, Ketones: (-).

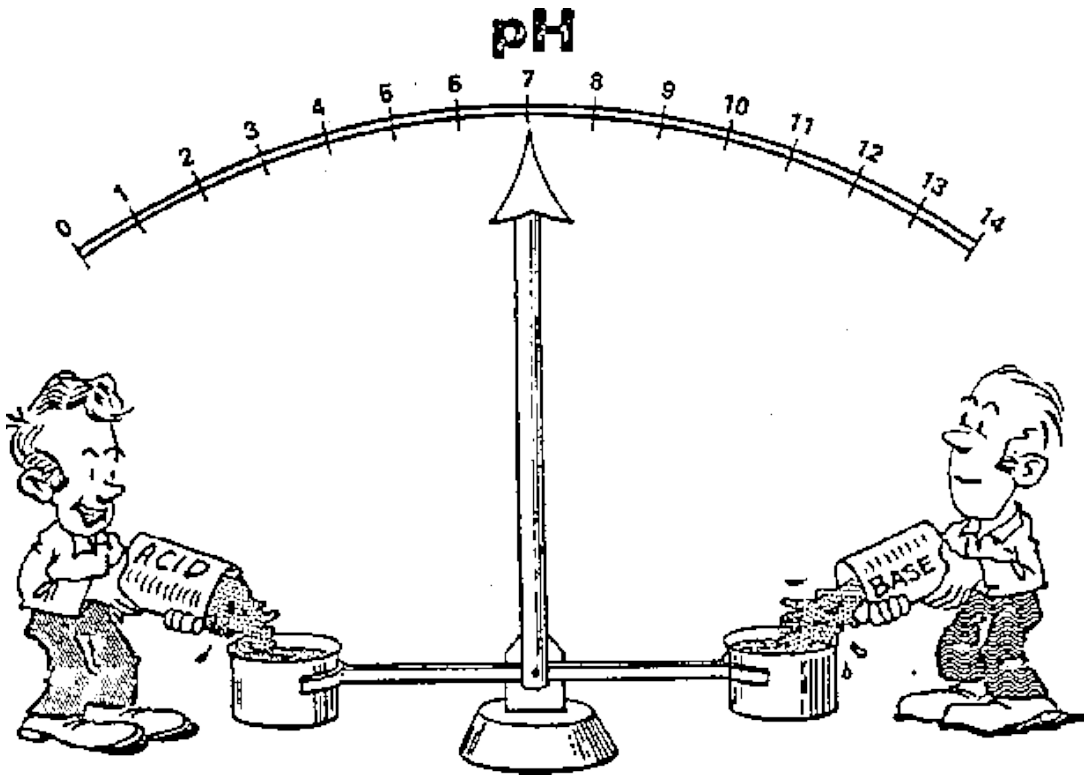
- Ποια η βασική οξεοβασική διαταραχή?
- Αν η ωσμωτικότητα του ορού μετριόταν σε 320 mOsm/L τι θα συμπεραίνατε?
- Πως θα ερμηνεύατε την ήπια αύξηση του γαλακτικού?

Ποια η βασική οξεοβασική διαταραχή?
Μεταβολική οξέωση
Expected PCO_2=(1.5 × HCO_3)+(8±2)=29±2 mmHg
AG=22 mEq/L
ΔAG/Δ[HCO_3] = 10/10 = 1

Αν η ωσμωτικότητα του ορού μετριόταν σε 320 mOsm/L τι θα συμπεραίνατε?
Υπολογιζόμενη ωσμωτικότητα: Ω= 2·[Na] + [Glu:18] + [Ur:6]=292 mOsm/L
Αυξημένο ωσμωτικό χάσμα πλάσματος (> 20 mOsm/L)
Πιθανή δηλητηρίαση με τοξική αλκοόλη (μεθανόλη, αιθυλενογλυκόλη)

Πως θα ερμηνεύατε την ήπια αύξηση του γαλακτικού?
Αυξημένη παραγωγή αναγωγικών ισοδυνάμων κατά το μεταβολισμό της τοξικής αλκοόλης από την αλκοολική αφυδρογονάση.


Glucose
PEP
Pyruvate
G6P
ATP
ADP
FBP
2 NAD+
2 ADP
+ 2 Pi
2 NADH
2ATP
2 (3-PGA)
2 PEP
2 ADP
2 ATP
2 Lactate
2 Pyruvate
2 NAD+
2 NADH



THANKS!