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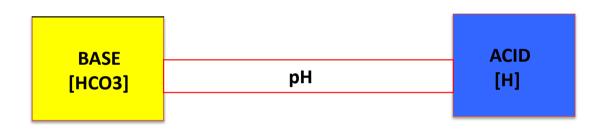
# Introduction to Acid Base Disturbances

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### **Outline**

- Components of Acid Base physiology
- Protective mechanisms that keep us alive
- How things can go wrong
- Acid Base interpretation with confidence
- Interactive cases

To measure the acidity in our system we do direct measurement of H concentration <u>But</u> that isn't practical so we measure the acidity **in**directly by measuring the pH which is suitable for the clinical settings.



#### Normal

рН	7.8	7.7	7.6	7.5	7.4	7.3	7.2	7.1	7.0	6.9	6.8
Н	16	20	26	32	40	50	63	80	100	125	160

# Henderson-Hasselbalch equation to calculate pH

Bicarbonate conc. We measure it directly,

$$pH = 6.1 + 1.3$$
  
 $pH = 7.4$ 

\* pH can never be less than 6.1 • pK = 6.1

carbonic acid

- HCO3/H2CO3 ratio = 20/1 (26/1.3)
- H2CO3=0.03×PCO2 and we use this equation to calculate the



#### **Exogenous**

- Physiological: Diet
   Any diet has amino acids
- Pathological: toxins (Methanol, Ethylene Glycol)

#### **Endogenous**

- Physiological: metabolism ( volatile & non-volatile acids)
- Pathological: Ketoacids and lactate

### **BASE**

Bicarbonate is the abundant base in our system

- HCO3 is the kidney favorite's player
- Liver produces HCO3 from some precursors

(Lactate, Citrate)

Patients with liver dysfunction will not be able to convert lactate or citrate to HCO3, they always at risk to develop some acidosis. As our body at a constant production of acid we need defense mechanisms

# Life saving mechanisms

- Blood Buffers Immediate front line, it's the <u>quickest</u> pathway and what keeps us alive to prevent extreme rapid deviation of pH (seconds)
- Respiratory reaction (ventilation) Intermediate (minutes)
- Kidney reaction (metabolic) Most efficient pathway (hours)

# **Blood Buffers**

Bicarbonate-Carbonic acid system How it's function?	53%
Hemoglobin	35%
Albumin	7%
Phosphate	5%

H2O + CO2 = H2CO3 = H + HCO3

Let's assume you have some free H ions that comes from your diet. Once it released every H ion will contained by bicarbonate (H+HCO3) and produce carbonic acid (H2CO3) which will dissociate to Co2 and water (H2O). CO2 will go out through the lungs and water either used in other metabolic pathway or goes out through the kidneys or the gut. This a very lifesaving mechanism, even if the H ion (acid) is not organic and from the other side of the equation when the respiratory chain from mitochondria produces a lot of Co2 which will bind to H2O and form carbonic acid (H2CO3) and dissociate by the carbonic anhydrase enzyme to H and bicarbonate which will repeat the cycle above.

# Respiratory mechanism

- Very quick reaction
- PCO2 and H have a potent stimulatory effect on the respiratory centre in the brain stem

### Renal mechanisms

By 2 things:

#### **Increase of HCO3**

- Absorption
- Generation

#### H acid secretion

• NH3 synthesis

Patient with kidney impairment have problems with these actions. That's why metabolic acidosis is common among those patients.

# Response to Acid load

Skipped by the doctor

- If 10 mmol/l of Acid is added to the blood
- pH = 6.1 + log ( Bicarb/carbonic acid)
   pH = 6.1 + log (26-10)/(1.3+10)
   = 6.1 + 0.15
   pH = 6.25 (if no protective mechanism exists)

# What can go wrong?

Most of acid base disturbances are not in blood buffer system

- Impaired respiratory response
- Impaired renal response

# Acid base interpretation

### Major tools in order to interpret with confidence

- pH
- [H] It can be calculated but we won't ask you to calculate hydrogen conc.
- [HCO3]
- PCO2
- Clinical data

You Never can interpret without the clinical info otherwise you will be subjected to a lot of errors.

#### **Supplementary tools**

#### The GAPs!

- Anion Gap What I need you to remember!
- Delta Gap
- Plasma osmolar Gap
- Urine anion Gap
- Urine osmolar Gap

# **Anion Gap**

What is it mean actually? It gives an idea about what you can't measure (غالبًا تكون أسيد)
So, if it's elevated tells you there's some extra acid that's you're unable to measure لكنه
ترا موجود بالبلود

- AG= Unmeasured anions Unmeasured cations
- AG= measured Cations measured anions
- AG = Na (Cl + HCO3)
- Elevated Gap indicates excess acids in the blood = Mhenever you calculate AG, check albumin! because big part of unmeasurable anions binds to albumin. So when it's low it may give a false image that there's no gab!
- Watch out for hypoalbuminemia!
- For each 10 point drop in albumin, add 2.5 to the calculated AG

تخيلوا الانيونز بالكفّة الأولى نايمة على عجينة ثقيلة من الالبومين (هذا الطبيعي) فلما يصير فيه اسيدوسيز وترجح كفّة الانيونز على الكاتيونز المفترض تزيد القاب صح؟ بحالة الهايبوالبونيميا هالشيء ما يصير بالعكس تروح العجينة وتتساوى الكفتين (مع ان الاسيد ارتفع!) وتقولين لا والله البيشنت حقي ما عنده اسيدوسيز ولا ارتفعت القاب! عشان كذا مع كل نقص ألبومين نزيد القاب نفس المكتوب فوق^

# Delta Gap mystery

 In metabloic acidosis, the drop in HCO3 should match the elevation in AG

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Delta gap= \Delta AG/\Delta HCO3 = 1
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- Delta gap < 1 = the drop in HCO3 is more than expected= 2 metabolic acidotic processes!
- Delta gap > 1 = the drop in HCO3 is less than expected= additional metabolic alkalotic process is present!

# Compensatory mechanisms Facts you should all know before continue

Acid base defect	Primary defect	рН	Compensation
Met acidosis	Low Bicarb	Low	Low PCO2
Met alkalosis	High Bicarb	High	High PCO2
Resp alkalosis	Low PCO2	High	Low Bicarb
Respiratory acidosis	High PCO2	Low	High Bicarb

# Normal Values

- pH= 7.4
- [H] = 40 nmol/l
- [HCO3] = 24 mmol/l
- PCO2 = 40 mmHg
- Anion Gap = 12

- Albumin = 40 g/l
- Delta Gap = 1
- Osmolar Gap < 10</li>

الكومبنسيشن تقدرون تستخدمون أي معادلة تبون مو شرط هذي لأن أصلًا كلها القاليديتي لك عليها

Acid base disorder	Primary defect	Compensation
Met acidosis	<b>⊎</b> нсоз	1.2 drop in PCO2 for each 1 mmol decrease in HCO3
Met alkalosis	<b>↑</b> HCO3	0.7 rise in PCO2 for every 1 mmol rise in HCO3
Acute resp acidosis	<b>↑</b> PCO2	1 mmol rise in HCO3 for every 10 point increase in PCO2
Ch resp aciodosis Chronic = >48h زي اللي عندهم COPD مثلًا	↑PCO2	3.5 mmol rise in HCO3 for every 10 point increase in PCO2 الكومبنسيشن صار أحسن لأن الكيدني هنا ص
Acute resp alkalosis	<b>V</b> PCO2	2 mmol drop in HCO3 for every 10 point fall in PCO2
Ch resp alkalosis	<b>₽</b> PCO2	4 mmol drop in HCO3 for every 10 point fal in PCO2

# Take the basic steps

- Describe the pH Acidemic or alkalemic
- Identify the primary drive for pH Is it low/high bicarb? Or low/high pCO2
- Predict the compensatory response
   Then after knowing respiratory or metabolic side you should know the compensation (calculate)
- Assess the actual compensatory response
- Calculate the Anion gap (AG)
- Correct the AG for albumin
- Calculate the Delta Gap (DG)
- Look for Osmolar gap (OG)

ونقارنها بالقيمة اللي عندنا ونشوف هل صار فيه أوقر كومبنسيشن والا اقل من المطلوب او بالضبط زي اللي حسبناه (هذا أساس الميكسد اسيد بيز بس ما راح أتكلم فيه بهالمحاضرة)

As a habit always calculate the AG and make the albumin corrections. DKA مرة مهم خصوصًا بحالات ال

# After reading a blood gas

- What is the primary disorder?
- Is it adequately compensated?
- Am I dealing with a single disorder or mixed disorders?

# What is the acid base disorder?

	pH (7.4)	PCO2 mmHg 10*1.2= 12 40-12= 28 (40)	HCO3 mmol/L (24)
A Acidemia:	<b>7.32</b> either high PCO2 or low HCO3	28 Adequately compensated	<b>14</b> Drop by 10 Metabolic acidosis
	<b>7.47</b> inical info to know is it acute or chron compensation	Respiratory alkaiosis	20
С	7.51	14*0.7= 9.8 40+9.8= 49 <b>49</b> Adequately compensated	<b>38</b> <sup>Elevated</sup> by 1 <sup>2</sup> Metabolic alkalosis
D	7.08	49	14
	Extremely low means No	Mixed we can't decide	Metabolic & respiratory Acidosis
	compensation	which complicated by which as we don't know the clinical	Acidosis

Even with compensation pH will **never** be normal it just to keep the cells alive.

info.

15 year old boy with abdominal pain,

рН	PCO2	HCO3
7.1	17	5 Drop by 19

19\*1.2= 22.8 40-22.8= **17** 

a. What is the acid base disorder?

<u>Adequately</u> compensated High anion gab Metabolic Acidosis

b. What else do we need to know?

We have to ask for Albumin!

Na 130 mmol/l, Cl 105 mmol/l

AG= 20

c. What is the clinical diagnosis?

DKA

If albumin is 15, From 40 to 15 there's 10 drop 2 times and a half so we add to AG 2.5 three times.

أي رقم بين ٢٠ ل٠٠ نضيف ٢٠٥

AG= 20+7.5= 27.5

23 year old man with a 3 day history of diarrhea.

#### ABG showed:

рН	HCO3	PCO2	
7.28	12	26	

Na= 135, Cl=110, K= 3.2 K is low because of diarrhea

Non-gabadequately compensated normal AG Metabolic acidosis

Cause of acidosis is the loss of bicarbonate because of diarrhea.

AG= 13 Must be corrected by albumin but let's say it's normal so this is the true AG=13

> #بالحسابات زيادة ونقص رقم أو رقمين ستِل أكسبتد

Always when the cause of acidosis is the loss of bicarb the AG will be **normal**! If it caused by accumulation of endo or exogenous acid it will be high.

Cases from now & on doctor said practice it by yourself

- 55 yo man k/c of BA. In ER with SOB and cough for 2 days
- ABG: pH= 7.32, PCO2= 50, HCO3= 25
- Na= 134 , K= 4.5 , Cl= 100

What is the acid base disorder?

- 55 yo man with COPD. Admitted for elective hernia repair. Pre operative ABG showed:
- pH= 7.37 , PCO2= 55 , HCO3= 31
- Na= 136, K= 3.5, Cl= 96

What is the disorder?

- 40 yo woman with repeated vomiting for 1 day. ABG showed :
- pH= 7.49 , PCO2= 48 , HCO3= 35
- Na= 130 , K= 2.8 , Cl= 85

What is the disorder?

28 yo man with abdominal pain and diarrhea.
 He is clinically volume depleted (low BP, tachycardia..)

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ABG:
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pH= 7.29 HCO3= 8, PCO2= 21

Na= 133, Cl=105

- Acidemia
- Metabolic acidosis
- Exp PCO2 = 20
- AG = 20
- ΔAG =8
- ΔHCO3 =16

Lactic acidosis)

Dx: combined Gap and non-Gap metabolic

acidosis (Diarrhea induced HCO3 loss and