Acid-base mysteries explained

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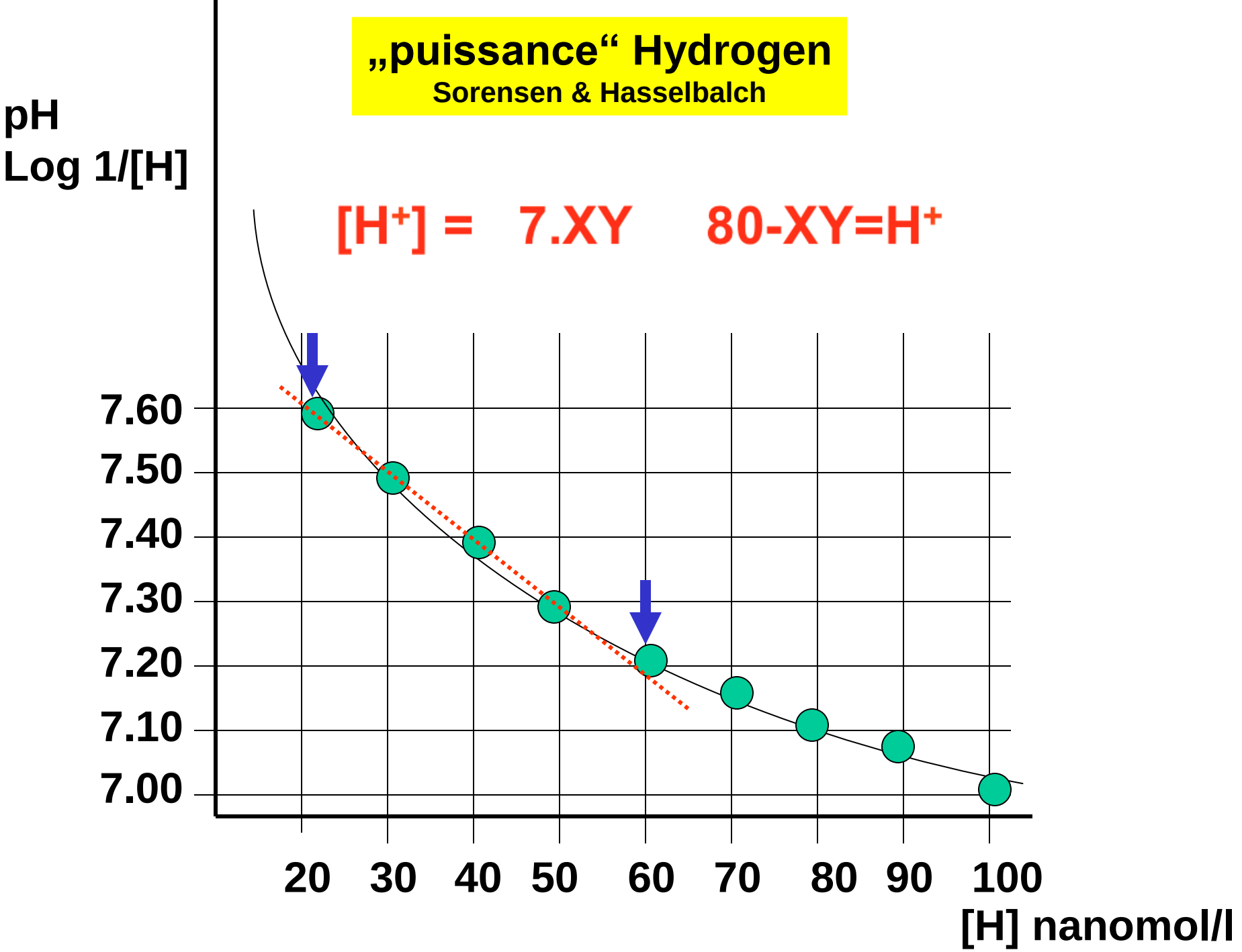
Hydrogen ion concentration pH 7.4

- **In plasma 40 nmol/l (compared to mmol for other electrolytes M, mmol, µmol, nmol)**
- **In cells about 60 nmol/l**
- **Concentration is expressed as pH (puissance hydrogen)**
- **pH = log1/H**
- **Water has pH 7.0 or $1x10^{-7}$ M or 0.000 000 1 M or 100 nmol/l**
- **Plasma has pH 7.4 or $1x10^{-7.4}$ or 40 nmol/l**

„puissance“ Hydrogen

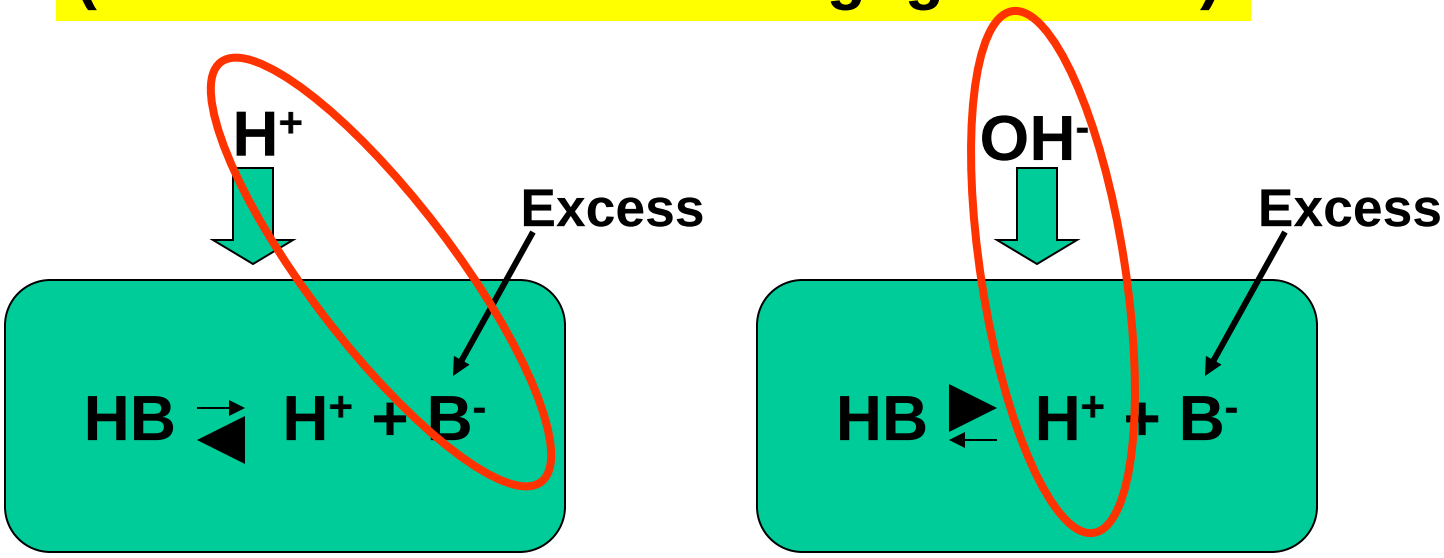
Sorensen & Hasselbalch



To convert pH to H (nmol/l) between pH values 7.20 and 7.60 subtract the last two digits behind the pH decimal value from 80

• 7.60 (pH)	20 [H] nmol/l	80-60=20	Flat part
• 7.50	30	80-50=30	
• 7.40	40	80-40=40	
• 7.30	50	80-30=50	
• 7.20	60	80-20=60	
• 7.10	90	extrapolate	
• 7.00	100	water=100	
• 6.90	125	seldom	

Buffer systems minimize pH changes: (Weak acid + excess congugate base)



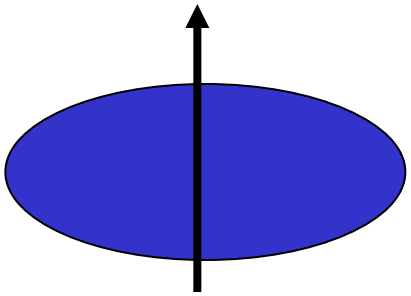
There are many buffer systems
H_2CO_3 + HCO_3 system provides about 60%
Hemoglobin provides about 35%
All the buffer systems are in equilibrium with each other

If we have to components to any one, we can calculate the pH relationships

HCO_3 system is an open system since we can manipulate both $PaCO_2$ and HCO_3

(HB) B^-

Alveolus: gasCO_2 ($PACO_2$ 38 mm Hg) 20 mmol/min



Alveolar capillary
dCO_2 ($PvCO_2$ 45 mm Hg; $PaCO_2$ 40 mm Hg)

Hb and other buffers

Solubility coefficieint

Liver from AA and urea cycle

1.3 mmol/l 40 nmol/l 24 mmol/l

$PaCO_2$ x 0.03 = H_2CO_3 H^+ + HCO_3
40 mmHg

$dCO_2 + H_2O$

Endproducts of metabolism 20 mmol/min

H^+ 80 mmol/d; NH_4Cl and fixed acids

Lawrence Joseph Henderson (* 3. June 1878 in Lynn, MS, USA; † 10. Februar 1942 in Cambridge) was an American chemist and biologist.

$$[H] = K \frac{H_2CO_3}{HCO_3} \quad \text{(K= Dissoziationskonstante)}$$

He wanted this:

$$H = \frac{24 \times pCO_2}{HCO_3}$$

(Henderson Formula)

$$[H] = 24 \frac{(pCO_2)}{HCO_3} = \frac{24 \times 40}{24} = 40 \text{ nmol/l}$$

Karl Albert Hasselbalch (* 1. November 1874 in Aastrup, Denmark; † 19. September 1962) was a physical chemist.

Henderson got this:

$$pH = 6.1 + \frac{\log HCO_3}{0.03 \times PCO_2}$$

Ever seen one of your staff solving this thing on the wards?

My anesthesiology friends are locked into jargon from the 1950‘s. They love and prosetylyze this stuff

Base excess (BE): The amount of acid or base that would be necessary, to titrate the pH value back to a value of 7.40.

Standard BE: Calculates the same assuming that the Hb = 5 g/dl.

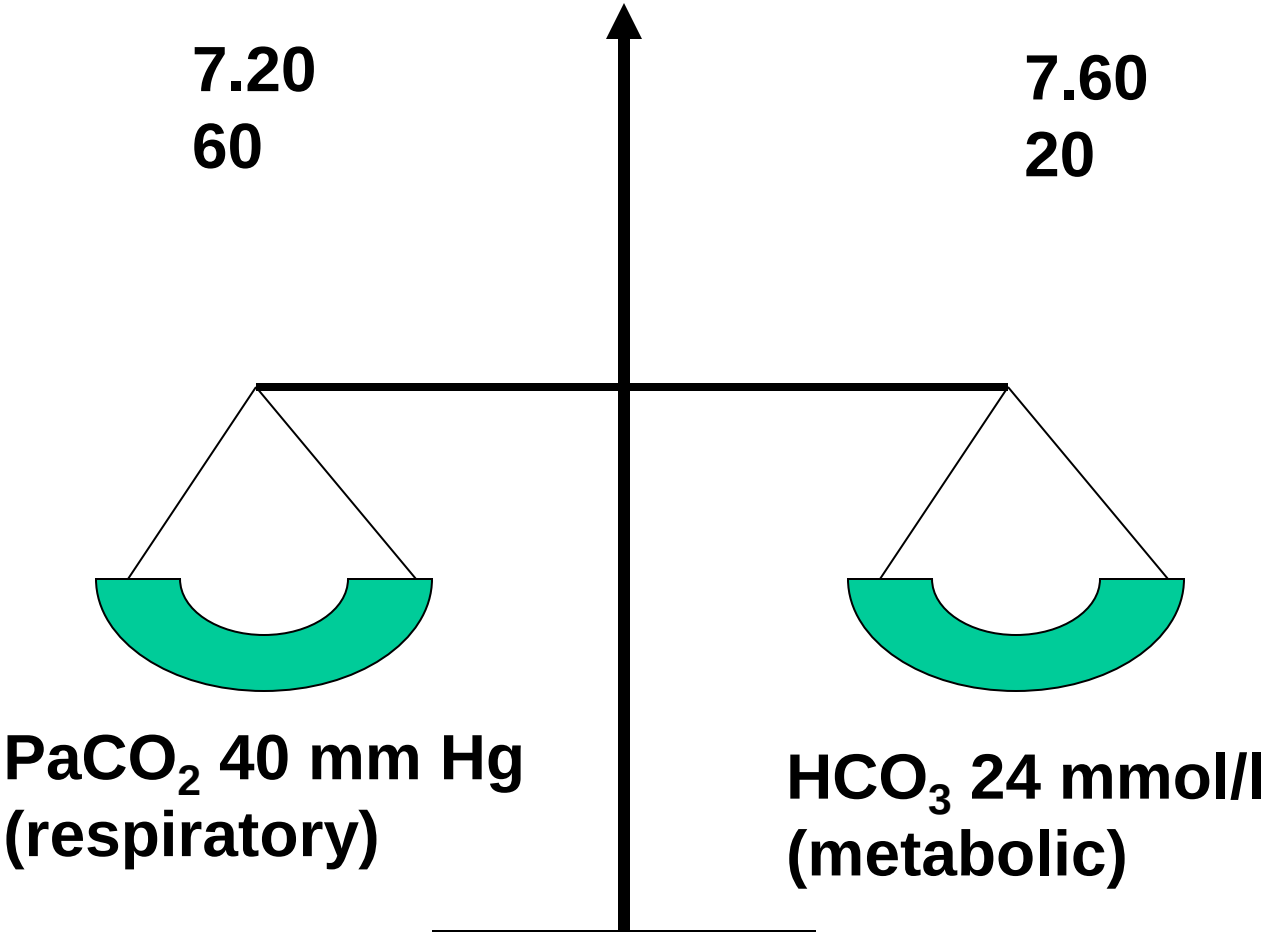
Standard pH (Hasselbalch): The pH under standardized conditions assuming: PCO_2 40 mm Hg,Temp 37 ° C, und O_2 100 % Sat.

Standard Bicarbonate: Defined as the metabolic component. It represents the HCO_3 value: were the PCO_2 40 mm Hg; Temp 37 ° C, und O_2 100 % Sat.

OK for 1950, but times have changed. Many physicians have not.

Acid-base „balance“

pH 7.4
[H] 40



Acidemia = pH<7.35
Alkalemia = pH>7.45

- Acidosis and Alkalosis are syndromes (disease)
- Metabolic acidosis or alkalosis is primarily a bicarbonate problem
- Respiratory acidosis or alkalosis is primarily a $PaCO_2$ problem
- If the primary disturbance is metabolic, then the compensatory response will be respiratory
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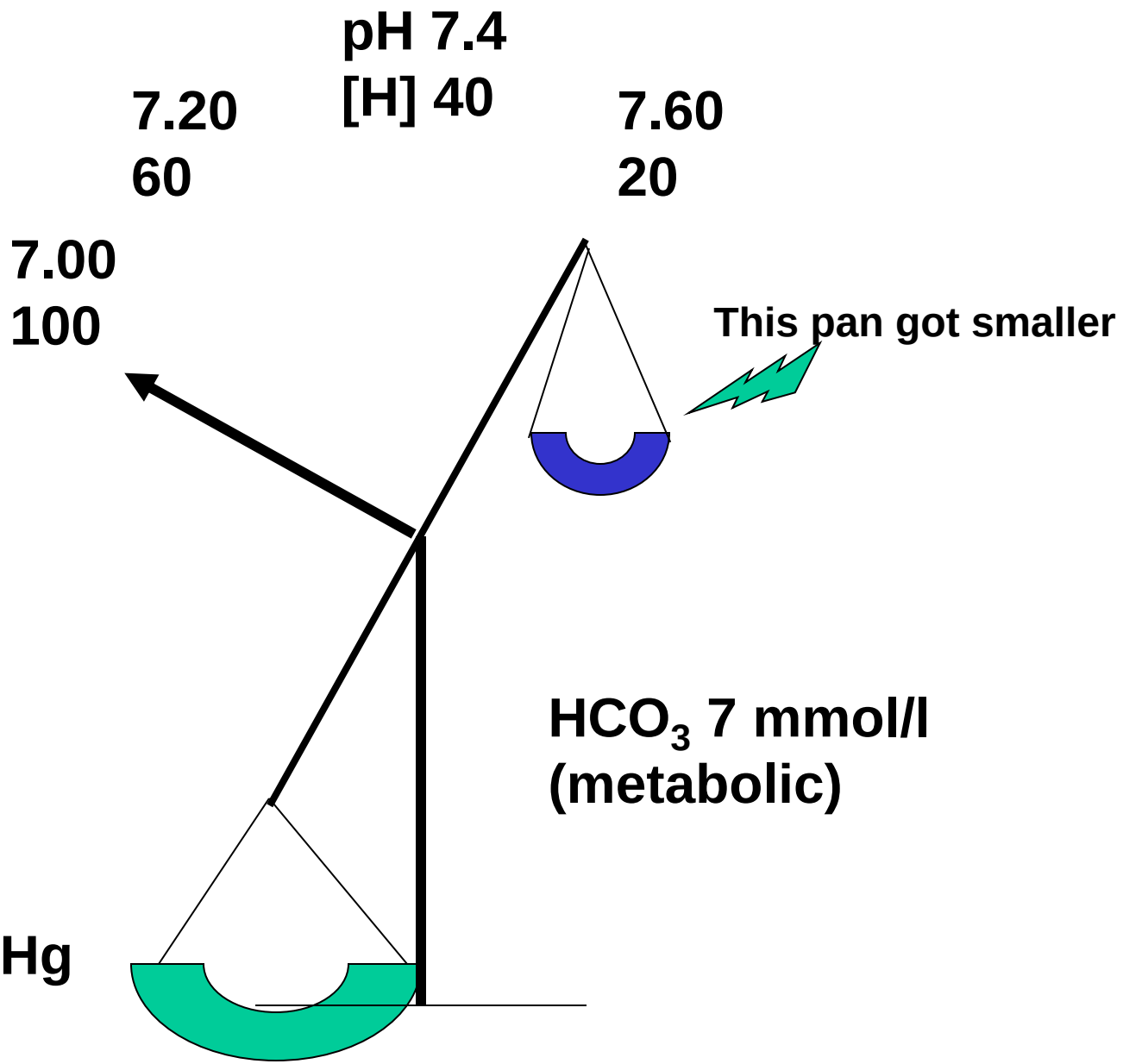
Acute metabolic acidosis



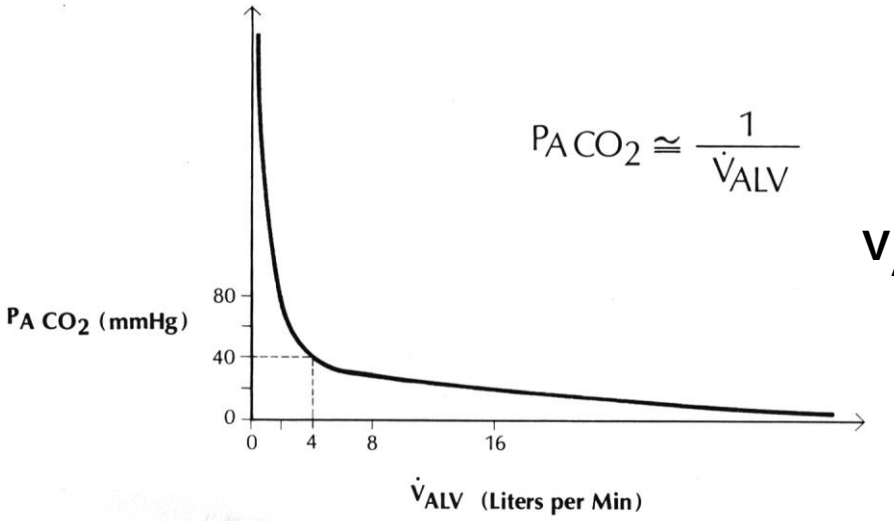
The respiratory component (H_2CO_3) is controlled by the lungs; we measure it as $PaCO_2$

The lungs excrete 20 000 mmol of CO_2 per day

The CO_2 content of a 70 kg person is 110 L

The $PaCO_2$ reflects the alveolar ventilation

$$ClCr = \frac{UCrxV}{SCr}$$



$$V_{ALV} \sim \frac{gCO_2 \times V}{\bar{P}CO_2}$$

28 year-old man, who 2 weeks ago developed polyuria, polydypsia, lethargy, and weakness. In emergency department, his temperature is 38° C.
BP is 90/50 mm Hg; HR 120/min; Resp 30/min

Patient #1

- **Creatinine 400 µmol/l (n<105)**
- **Urea 40 mmol/l (n<9)**
- **Na 137 (norm 136-144), Cl 105 (n 95-105), K 4 (n 3.5-5.2) (mmol/l)**
- **Glucose 30 (n<11) mmol/l**
- **Urine stick Ketone ++++**
- **pH 6.97, $PaCO_2$ 18 mm Hg, PaO_2 125 mm Hg (room air), HCO_3 4 (n24) mmol/l**

What could kill this patient within the next hour or so? You are offered his pH, his glucose, his bicarbonate, his creatinine, his failure to embrace the correct religion, his volume-depletion (shock)?

Acute metabolic Acidosis

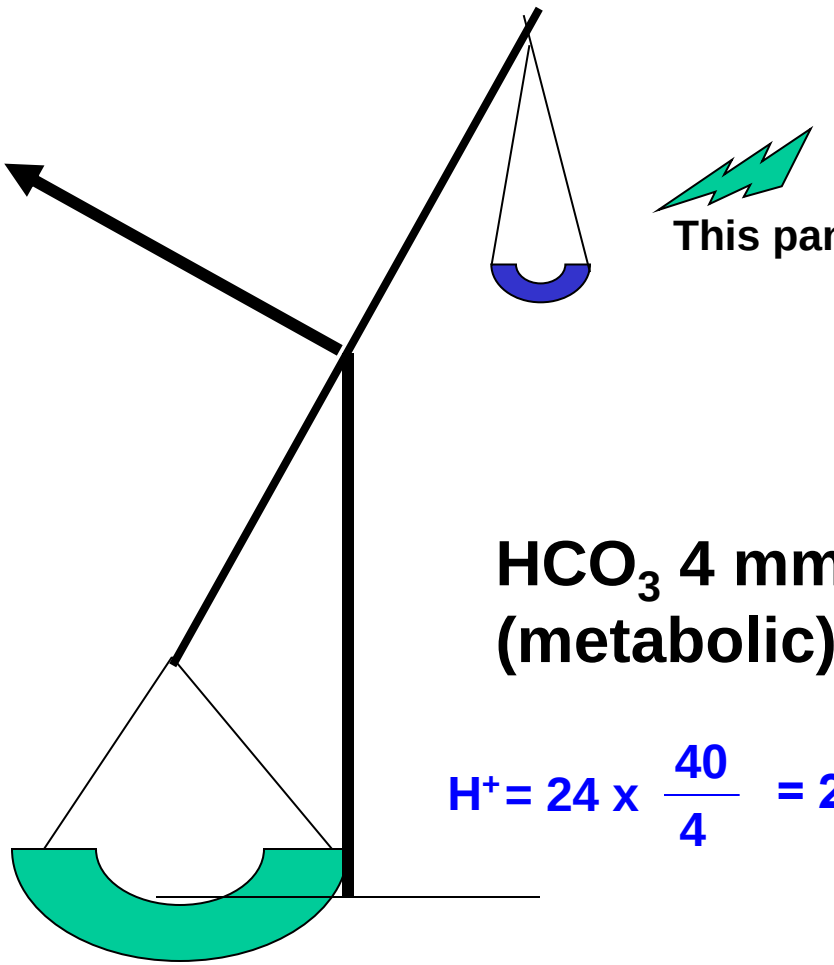
$H_2O + CO_2 \longleftrightarrow H_2CO_3 \longleftrightarrow H^+ + HCO_3^-$

7.20
60

pH /.4
[H] 40

7.60
20

7.00
100



This pan collapsed

HCO_3 4 mmol/l
(metabolic)

$H^+ = 24 \times \frac{40}{4} = 240$ nmol/l ≈ pH 6.62

$PaCO_2$ 40 mm Hg
(respiratory)

Respiratory compensation comes within minutes

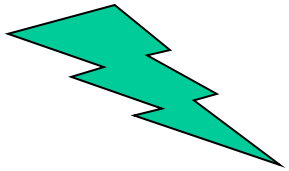
$H^+ = 24 \times \frac{18}{4} = 108$ nmol/l ≈ pH 6.97

pH 7.4
[H] 40

7.20
60

7.60
20

This pan is actively reduced



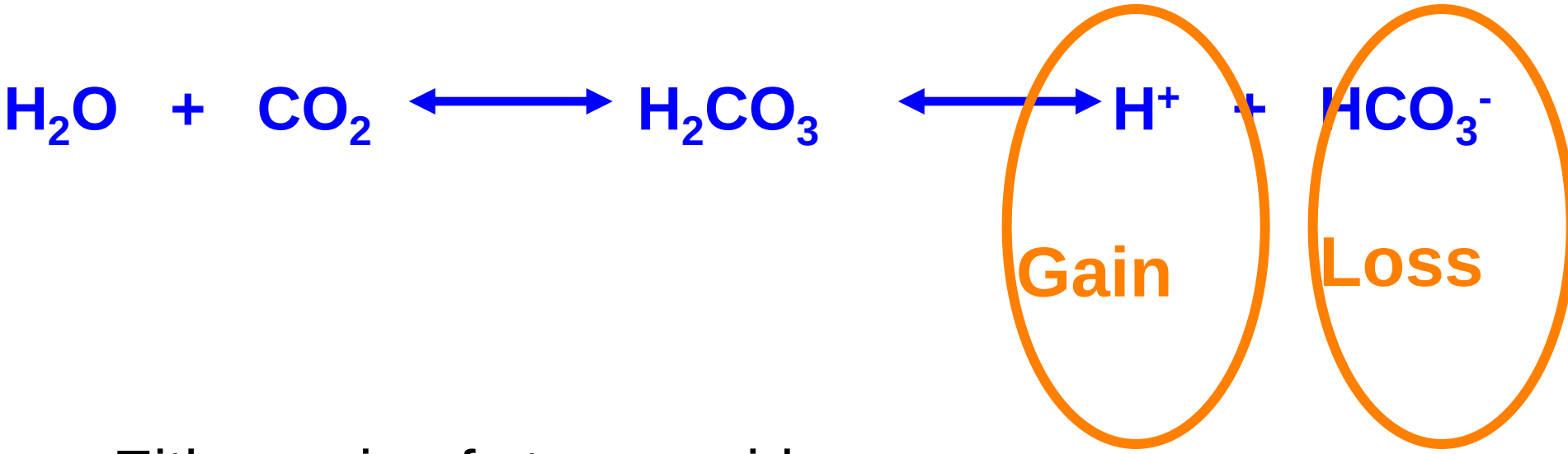
- 1 mm Hg $PaCO_2$

- 1 mmol/l HCO_3

HCO_3 4 mmol/l
(metabolic)

$PaCO_2$ 18 mm Hg
(respiratory)

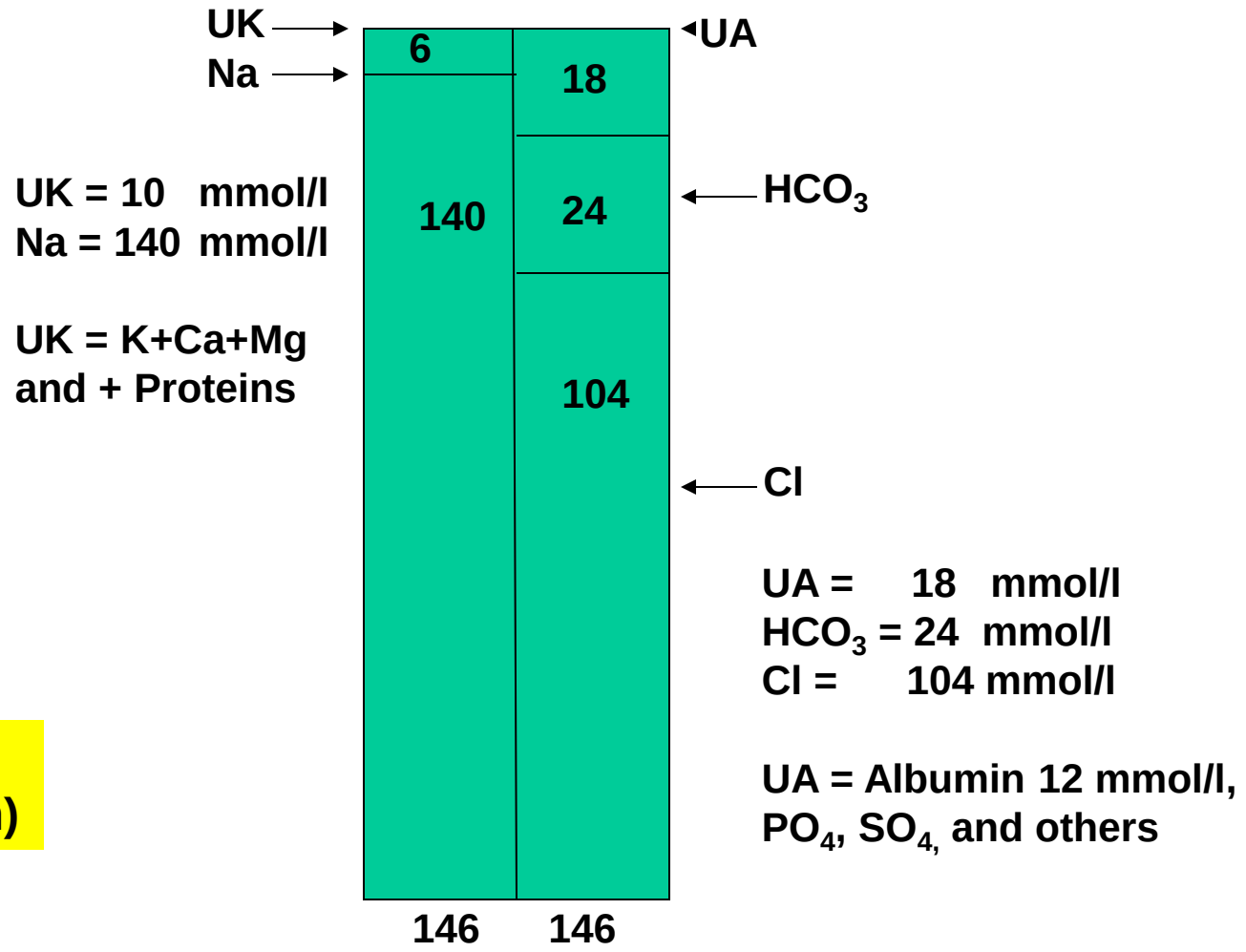
Metabolic acidosis is a HCO_3 problem; only two mechanisms can be responsible



- Either gain of strong acid
  - In that case the, H^+ require an accompanying anion.
  - HCO_3 be lost from the body.
  - Then we need to find out where the HCO_3 could be going.

The „metabolic component is the HCO_3 Ion in plasma; The cations = anions in terms of their electrical charge meq/meq (mval/mval).

James Lawler Gamble (Pediatrician from Boston)



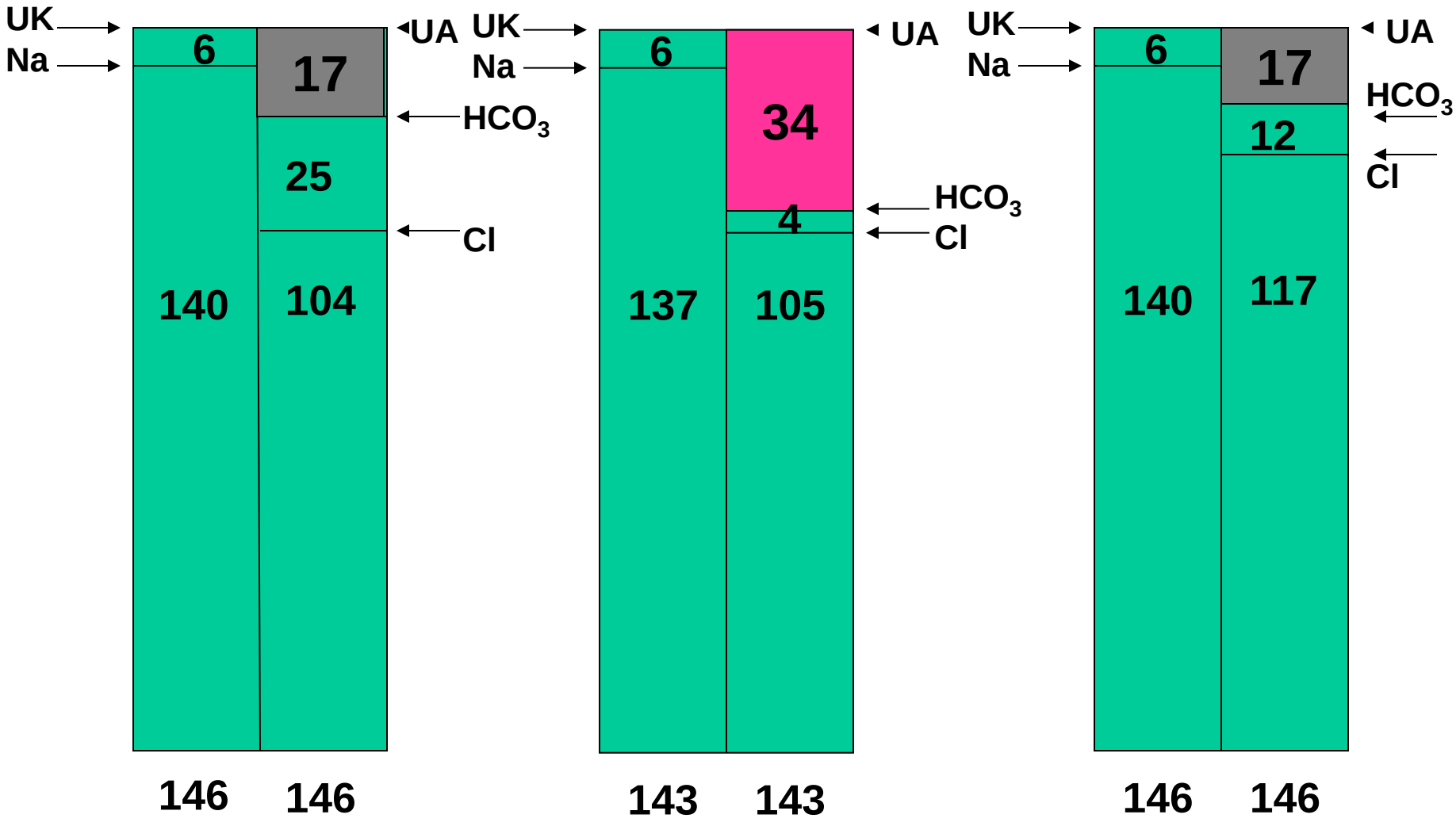
UK = 10 mmol/l
Na = 140 mmol/l

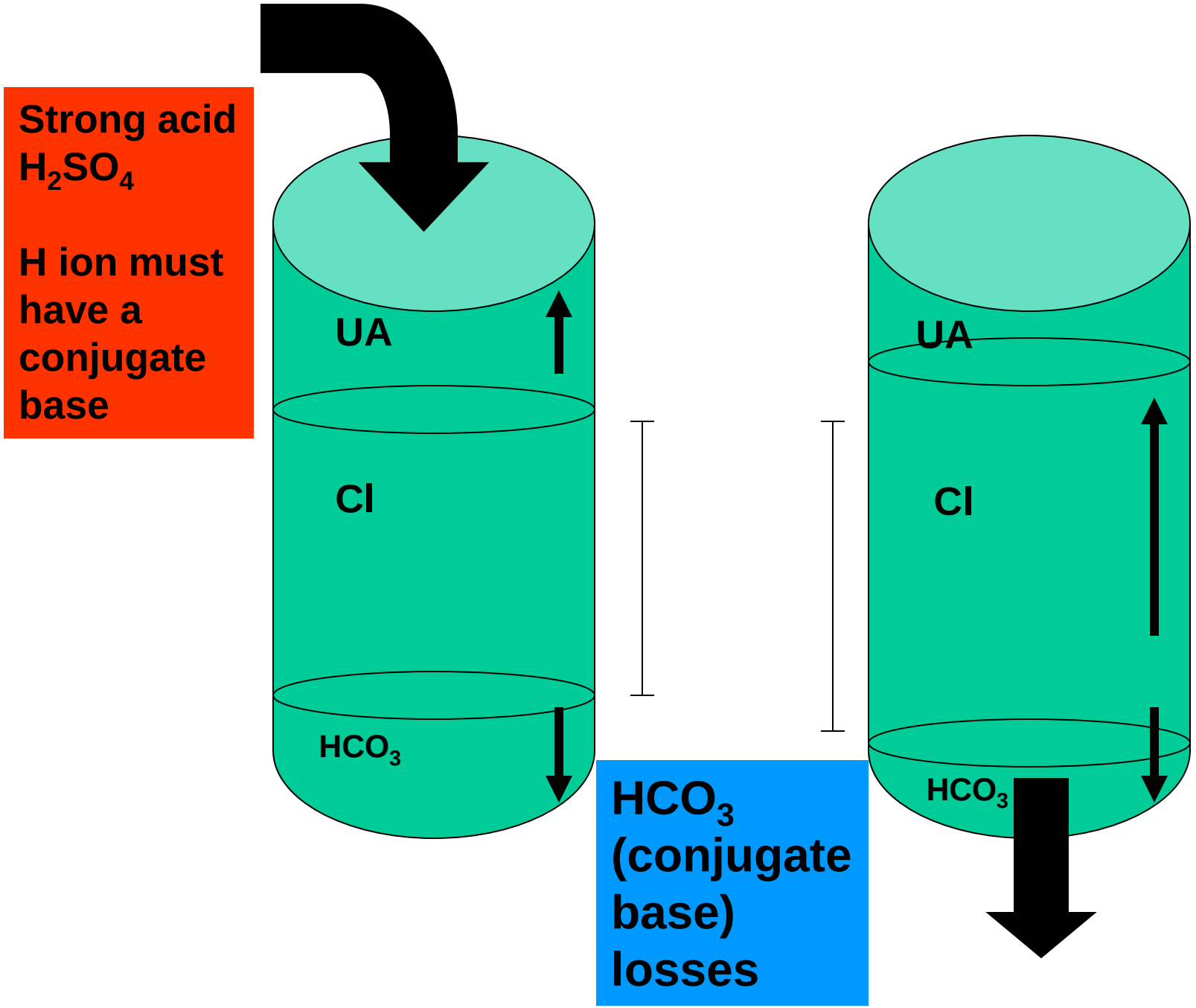
UK = K+Ca+Mg and + Proteins

UA = 18 mmol/l
HCO_3 = 24 mmol/l
Cl = 104 mmol/l

UA = Albumin 12 mmol/l, PO_4, SO_4, and others

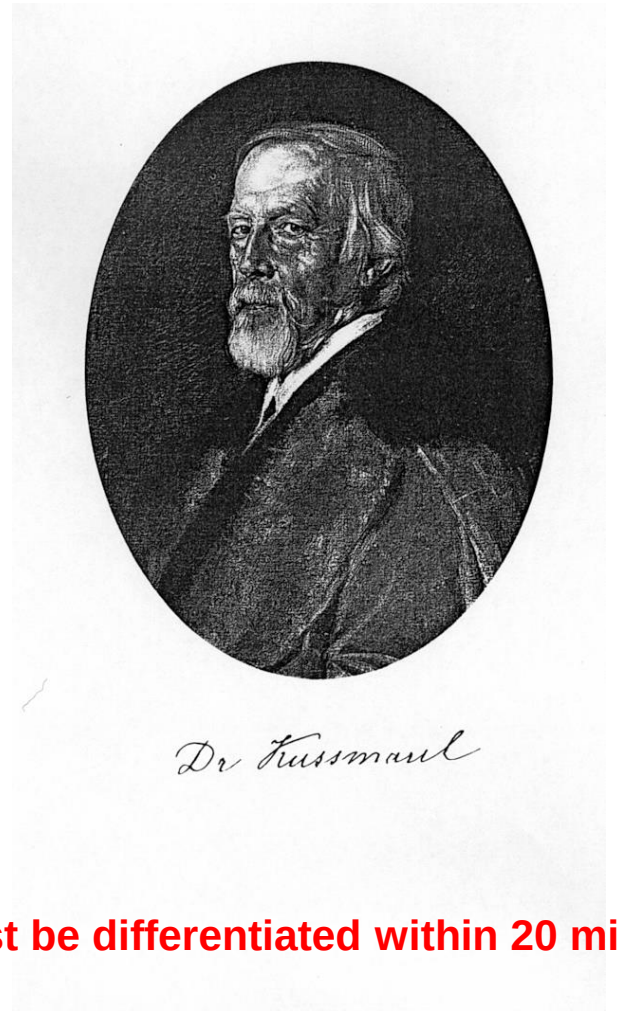
Metabolic acidosis with increased anion gap




Strong acid
H_2SO_4
H ion must have a conjugate base
UA
Cl
HCO_3
UA
Cl
HCO_3
HCO_3 (conjugate base) losses

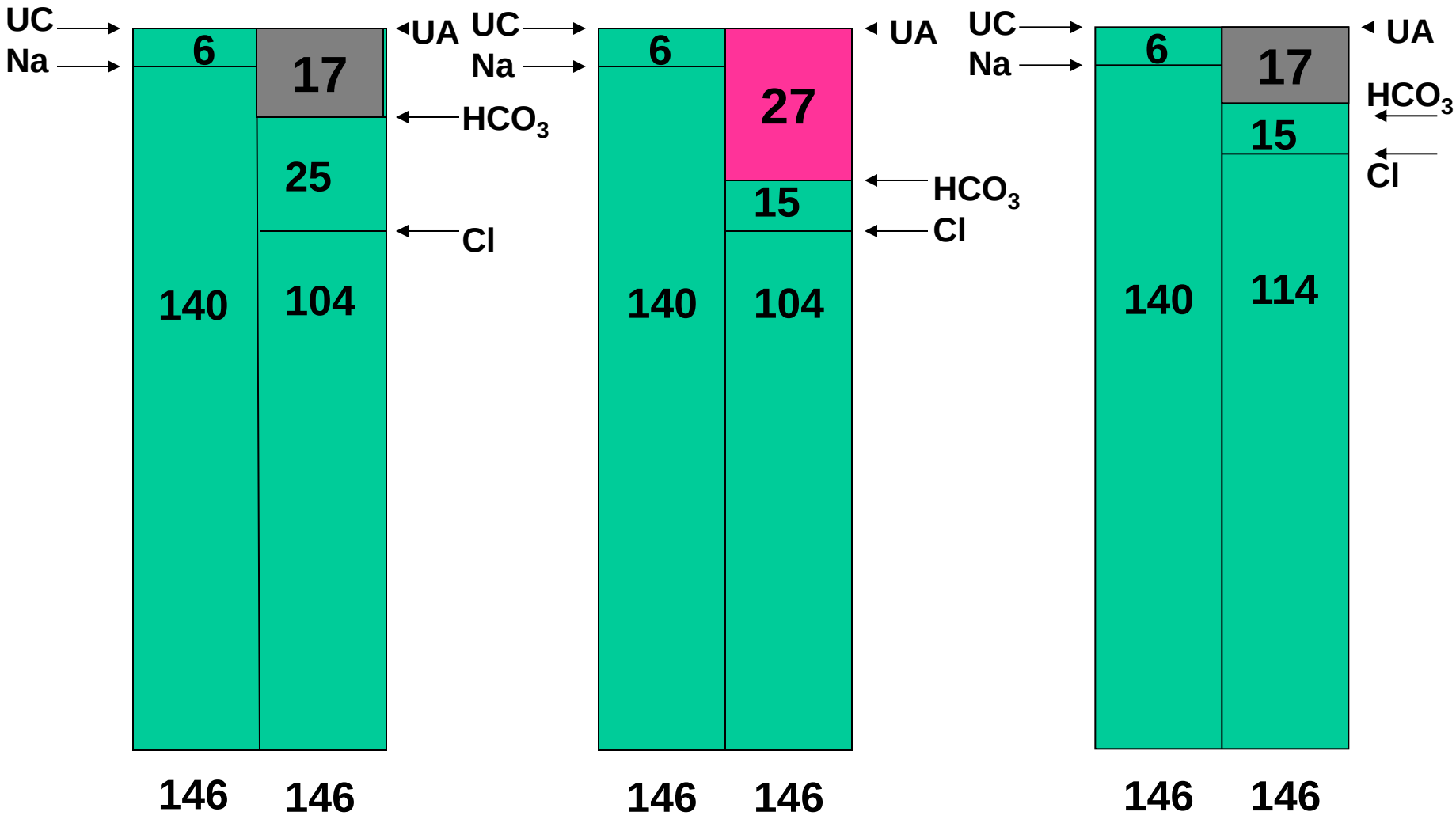
Wide anion gap after Adolph Kussmaul

- **K**etoacidosis
- **U**remia
- **S**alicylic acid (aspirin)
- **M**ethanol
- **E**thylene glycol
- **U**remie (zwei mal für Anfänger)
- **L**actic acidosis

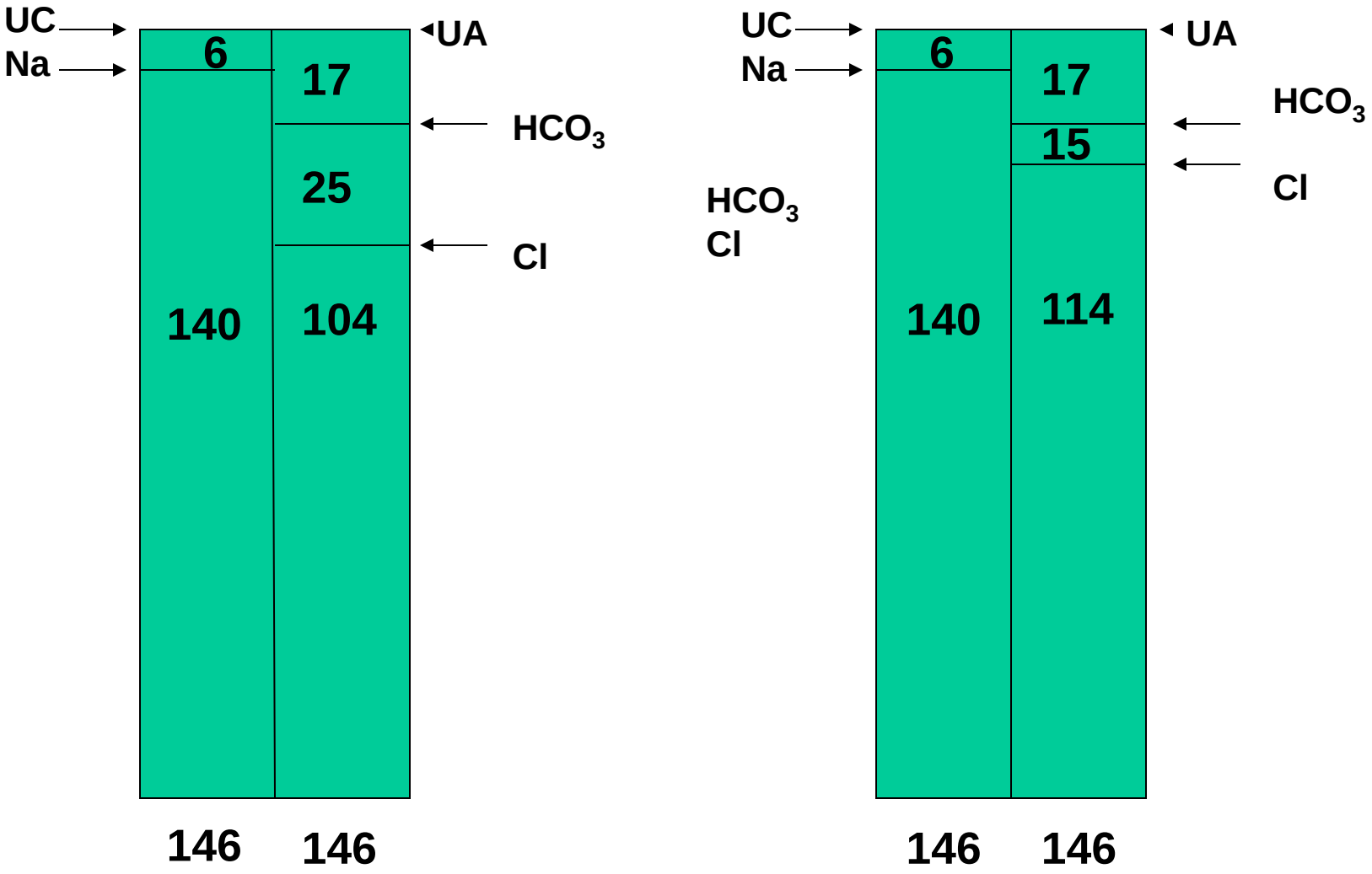


There are very few others; the concept counts. These must be differentiated within 20 min.

Metabolic acidosis with increased and nonincreased UA



Metabolic acidosis with a non-increased UA: Where did the HCO_3 go???

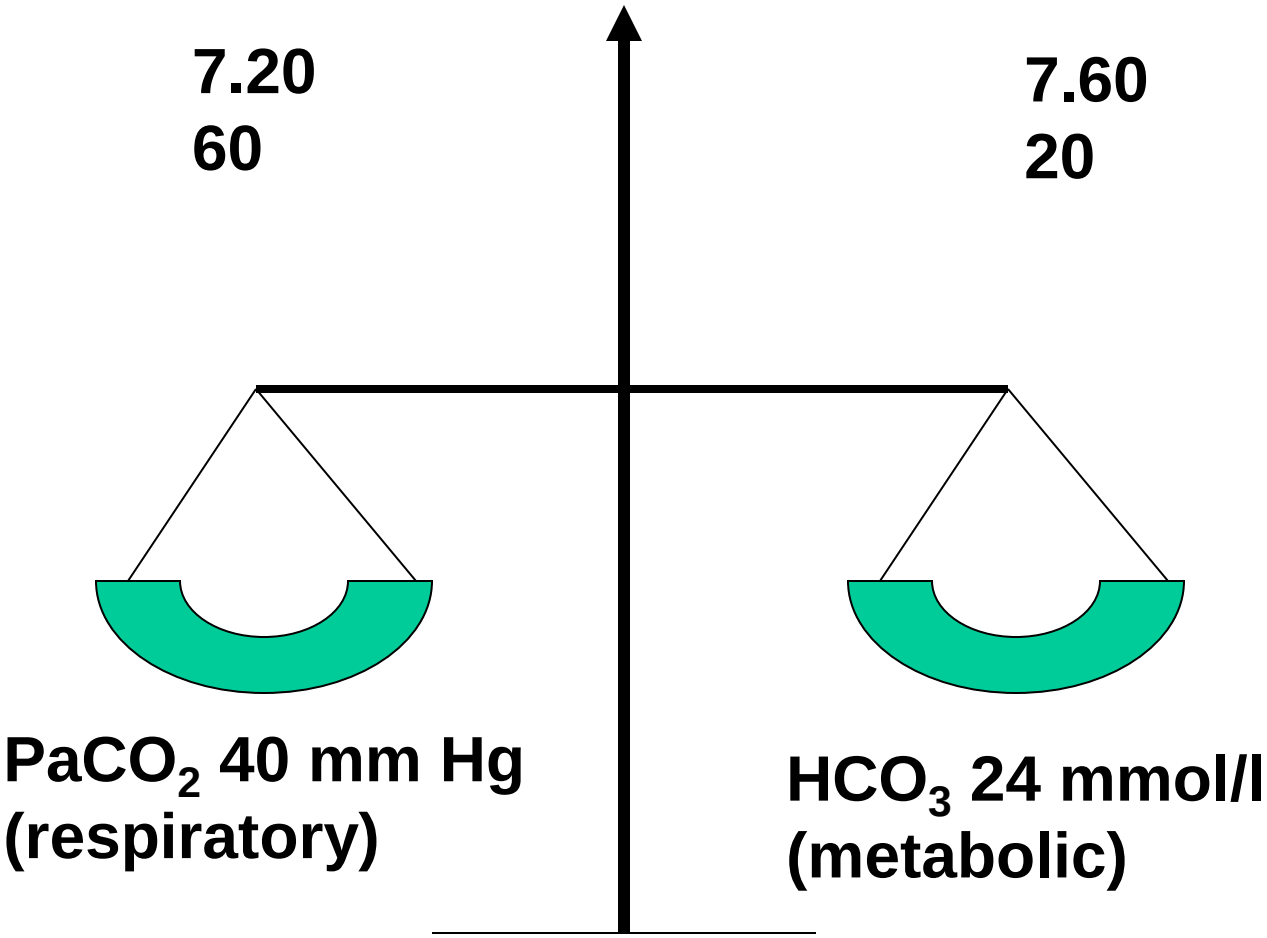


How does one lose HCO_3 ??
(Count the holes: Try lower GI tract or kidney)

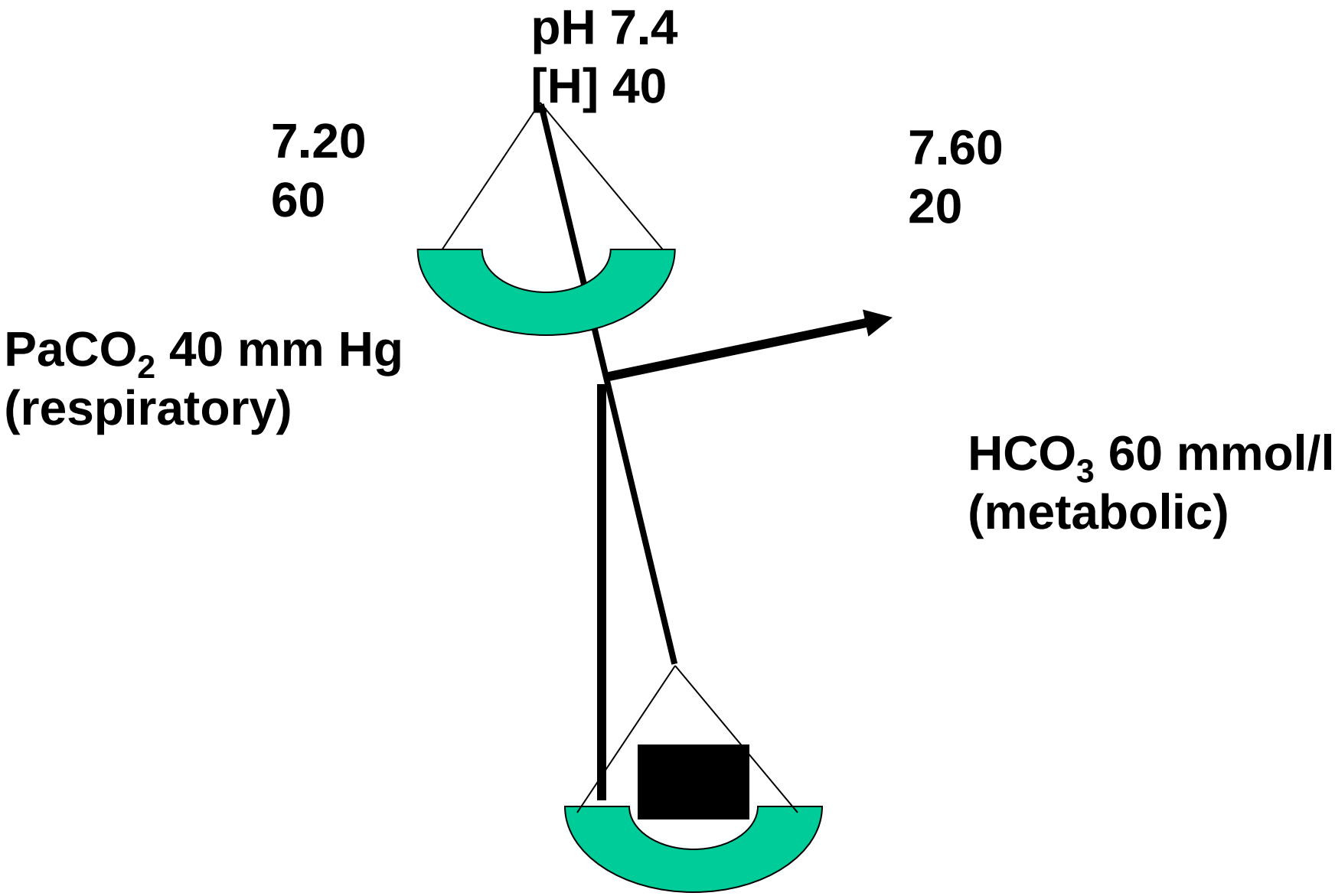
- How does one lose HCO_3 ??
- (Count the holes: Try lower GI tract or kidney)
- Diarrhea
- Pancreatico-Cutaneous fistula
- Gall bladder drainage
- Kidney cannot reabsorb bicarbonate (proximal)
- Kidney cannot excrete hydrogen ion (distal)
- Not enough kidney left to excrete NH_4 (chronic renal failure)

Acid-base „balance“

pH 7.4
[H] 40



Acute metabolic alkalosis: how can this happen?

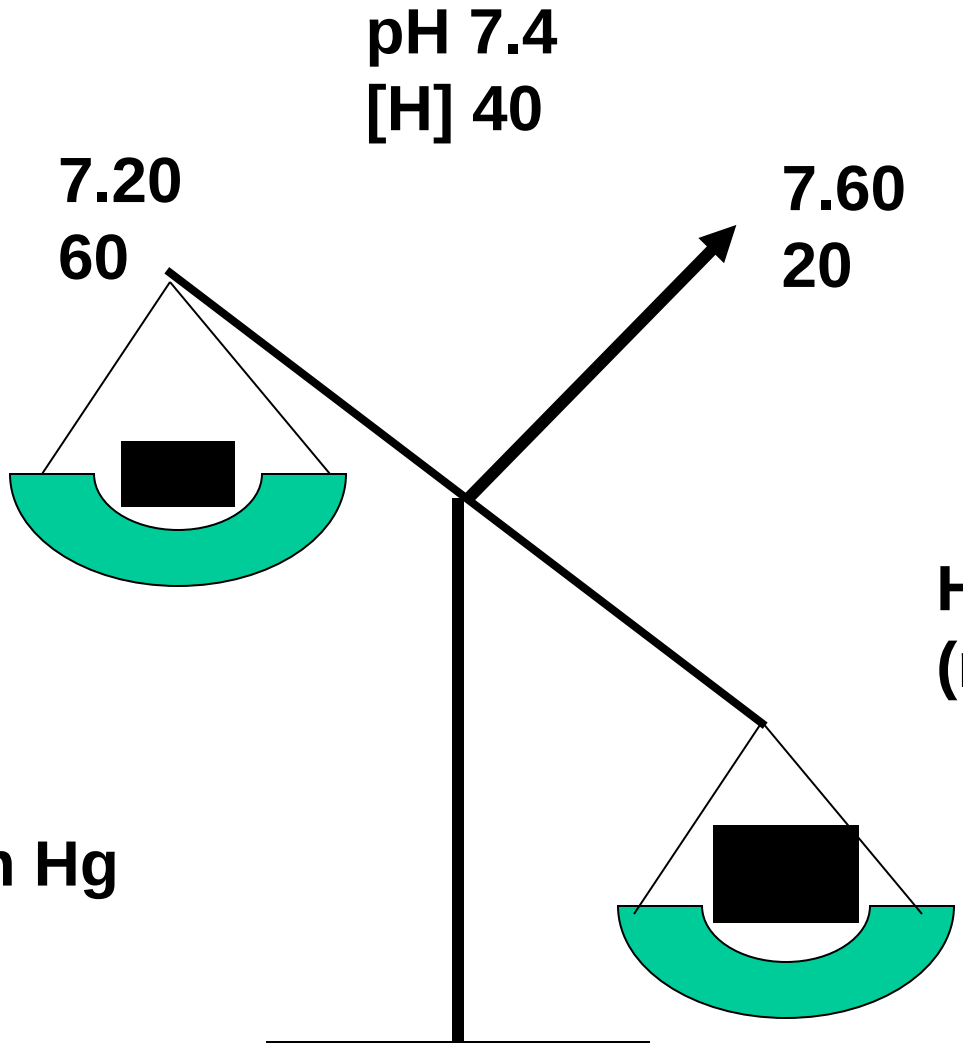


Case: A 56 year-old alcoholic woman was admitted with "several days" of abdominal pain with vomiting. She had a history of ulcer disease and pancreatitis. She smoked 2 packs daily since age 12 yr. She had lost 10 kg. BP 100/70 mm Hg, HR 100/min RR 8/min, abdominal tenderness, EKG long QTc with "U" waves

- **Na 125, K 2.6, Cl 54, HCO_3 60 (mmol/l), pH 7.61, $PaCO_2$ 70, PaO_2 65 (mm Hg)**
- **PCr 7 mg/dl, BUN 80 mg/dl, Blood sugar 65 mg/dl**
- **Amylase elevated**
- **Gastroscopy, duodenal ulcer with pyloric narrowing**

- **Primary disturbance ?**
- **Compensatory response ?**
- **Hypokalemia ?**
- **Urinary electrolytes ?**
- **Treatment ?**

Compensated metabolic alkalosis: what are the penalties?



HCO_3 60 mmol/l (metabolic)

$PaCO_2$ 60 mm Hg (respiratory)

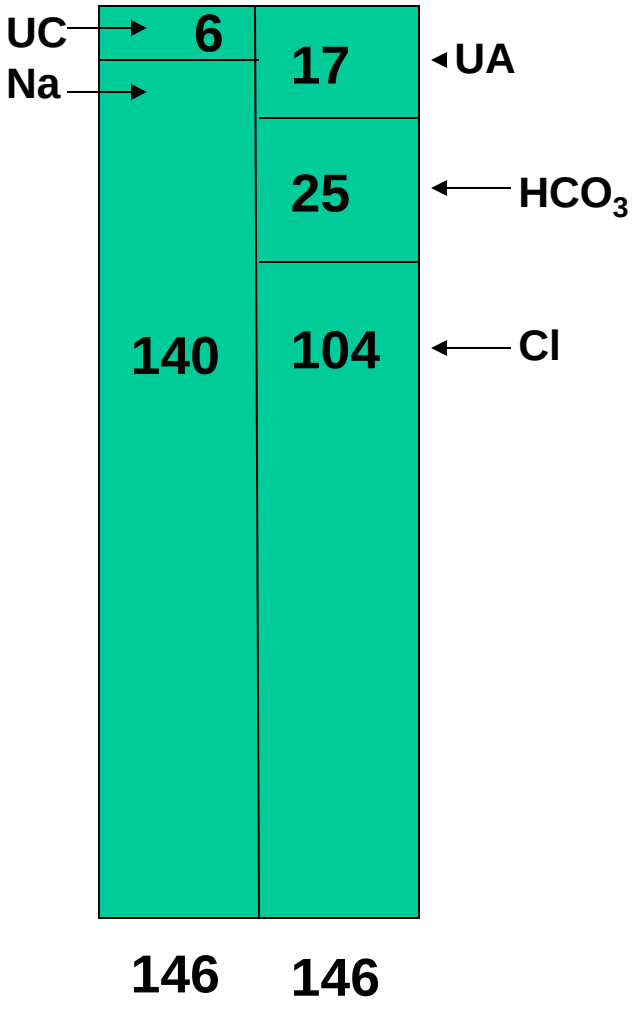
PaO_2 68 mm Hg

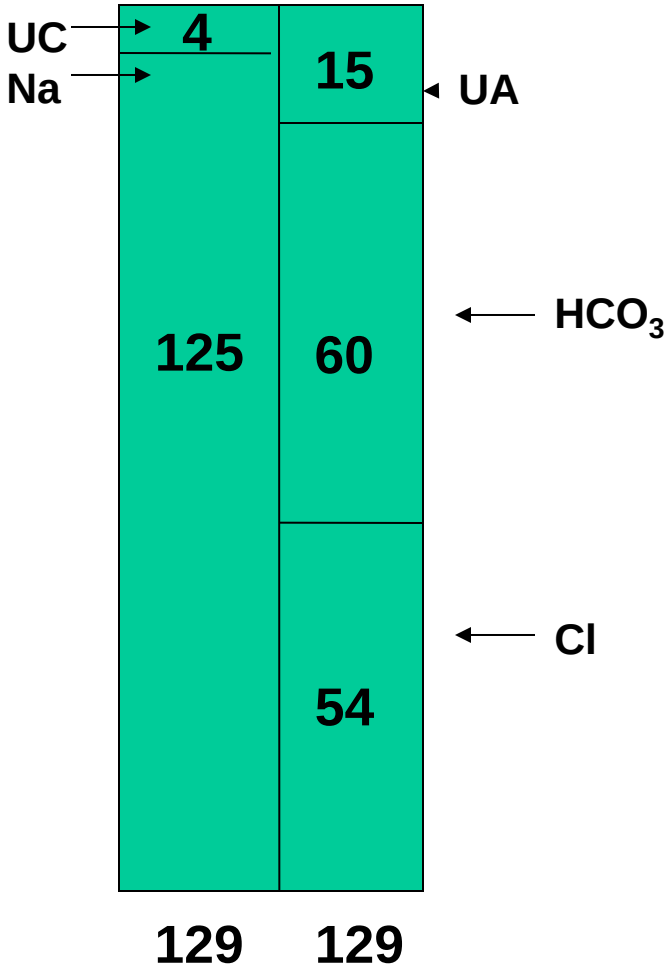
Metabolic alkalosis (a HCO_3 problem) can come about in only two ways

- H^+ can be lost from the body
  - In this case, the H^+ has an accompanying anion, namely chloride
- HCO_3 can be gained inappropriately
  - Some crazy people actually eat HCO_3 !!
  - HCO_3 may be gained because H^+ is being excreted by the kidney in exchange for Na reabsorption (mineralocorticoid effect)

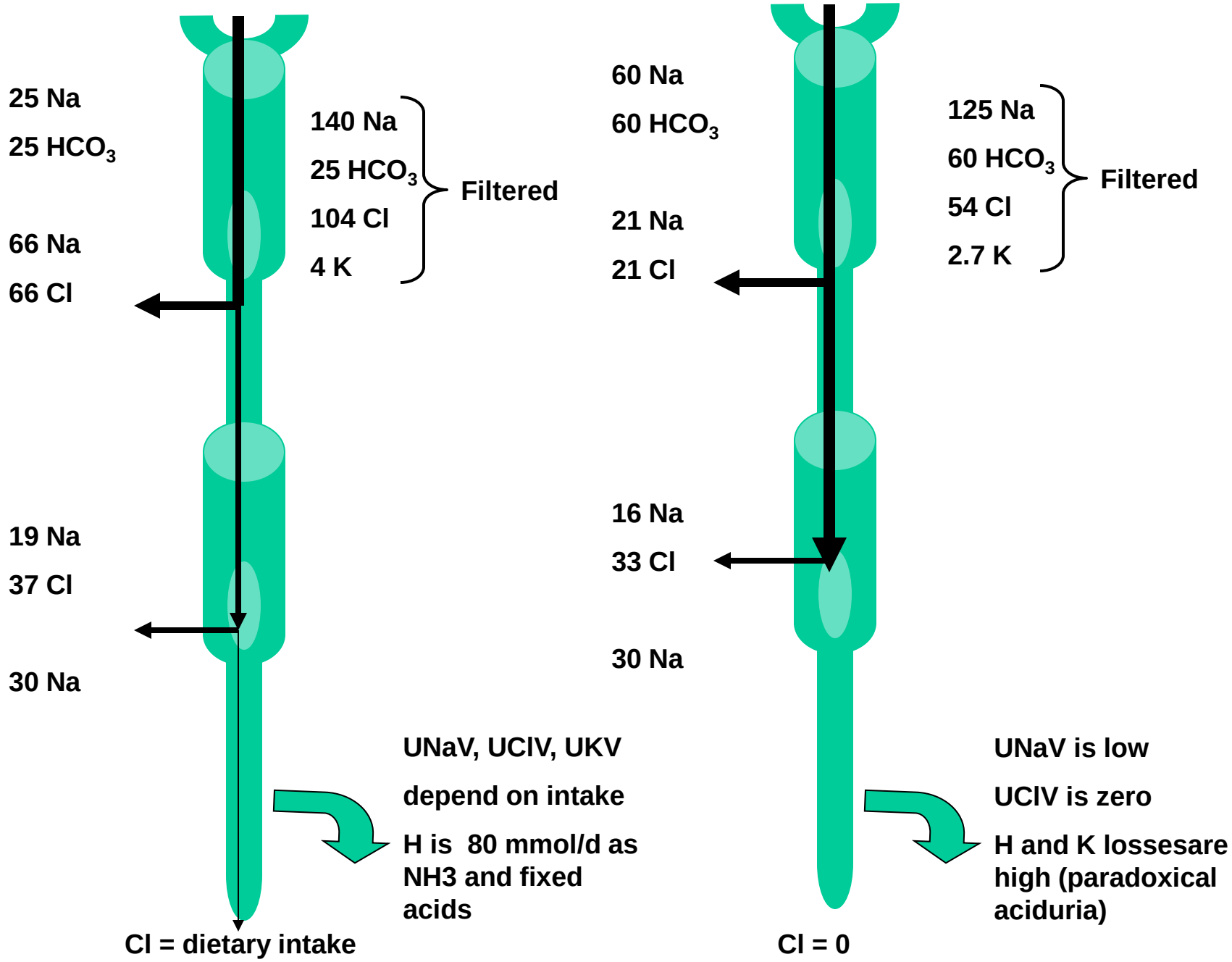
How must this patient reabsorb Na? Only 54 mmol/l

Can be reabsorbed as NaCl, the rest must rely on H exchange





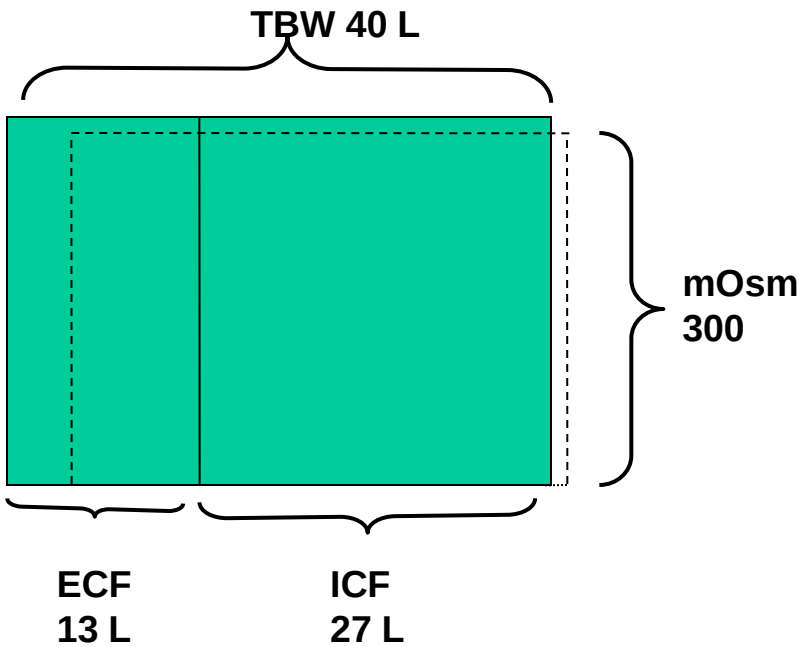
Why metabolic alkalosis is self-sustaining until the Cl deficit is replaced



How do you know that this patient does not have underlying chronic respiratory acidosis?

- What is the appropriate compensation for metabolic alkalosis ?
- Is the ECF volume contracted - and if so, why ?
- Is the renal response appropriate ?
- How can I test for chronic lung disease in this patient ?
- What is the alveolar-arterial O_2 gradient ?

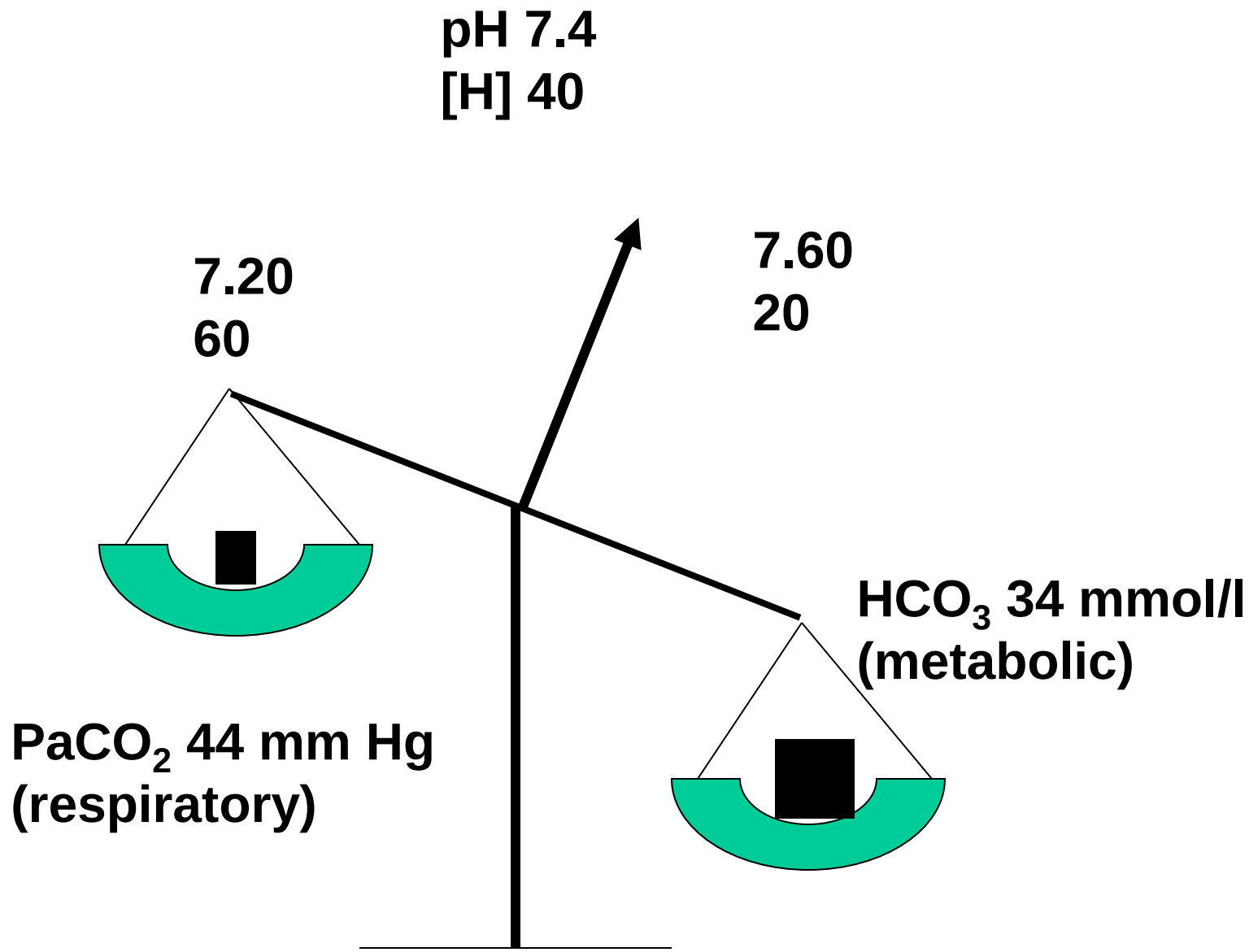
Patient had protracted vomiting



BP 100/70, HR 100
Na 125, K 2.6, Cl 54, HCO_3 60 (mmol/l),
PCr 7 mg/dl, BUN 80 mg/dl, Sugar 65 mg/dl
mOsm = 2(125+2.6) + 65/18 + 80/2.8 = 280

UNa= 10, UK 37, UCl = 1 (mmol/l)
Uosm = 450
TTKG = U/P K / U/P Osm
TTKG around 7
PAO_2=150-(60x1.25)=75
A:a 75-68=7 mm Hg
No lung disease

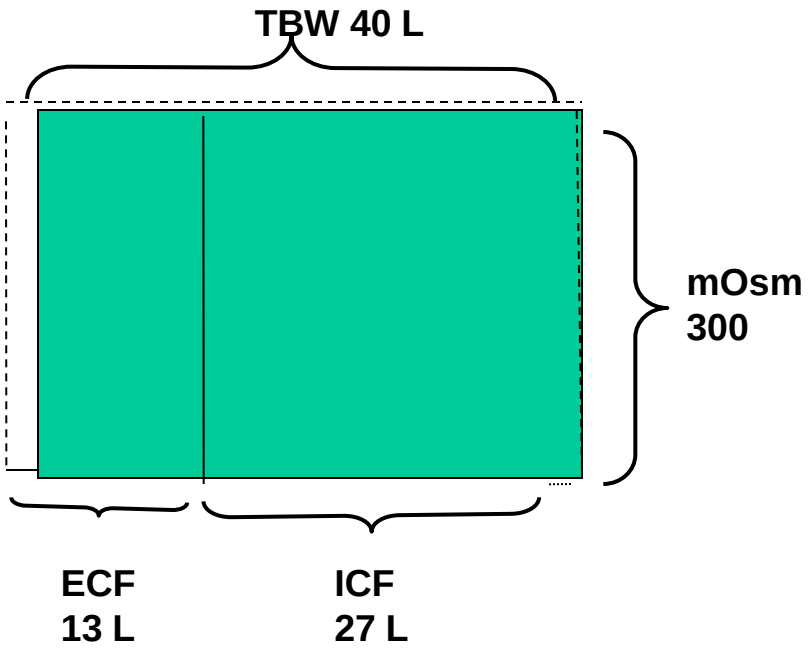
Chronic compensated metabolic alkalosis



Volume expansion with metabolic alkalosis;
No hypotension, no hypochloremia, yes hypertension;
Patients usually have a serum Na on the „high“ side of normal

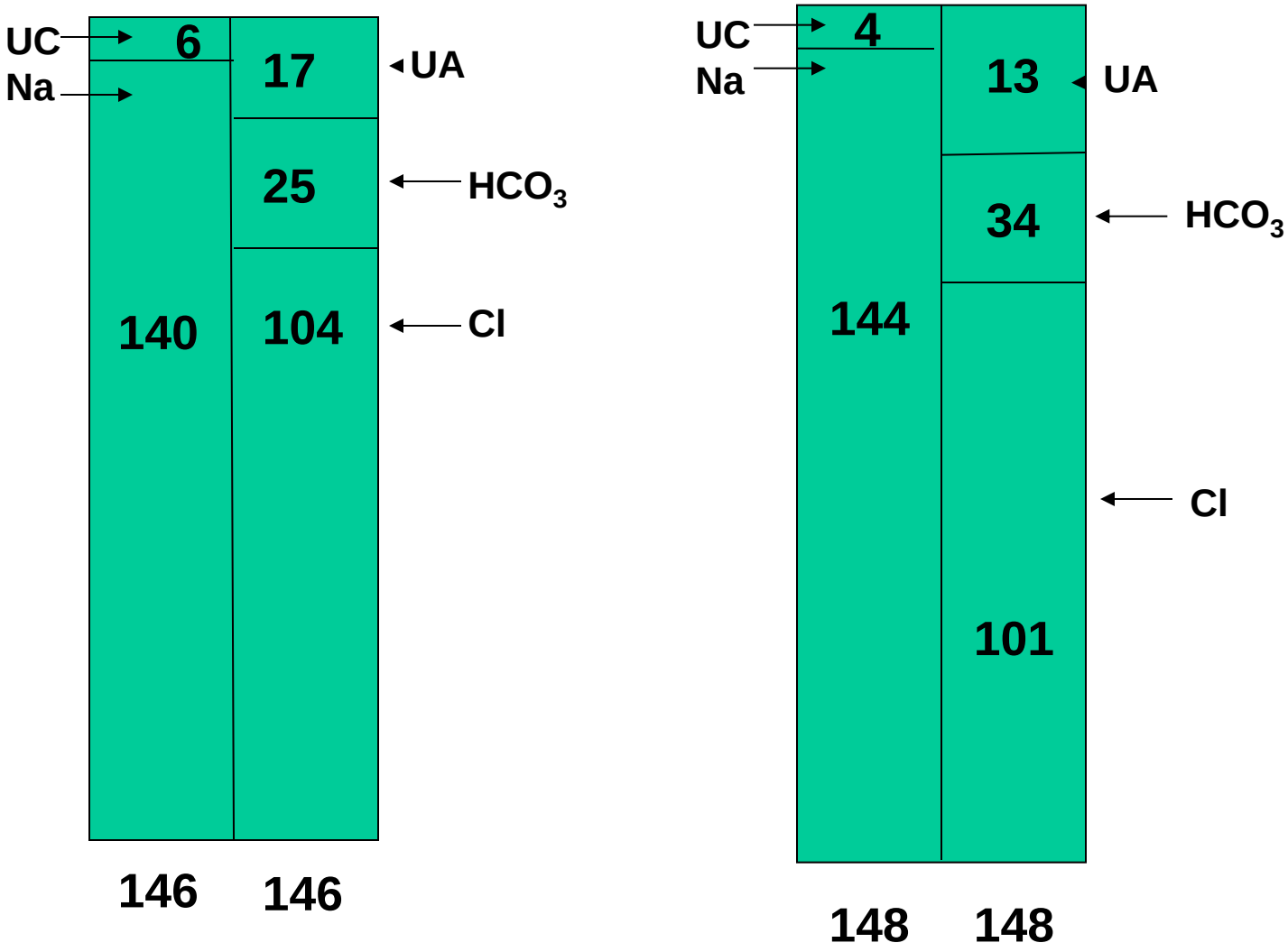
BP 150/110 mm Hg

Na 144 mmol/l
Cl 101
HCO_3 34
K 3.2



Patient had primary aldosteronism

Normal electrolyte relationship (left) compared to our patient (right)

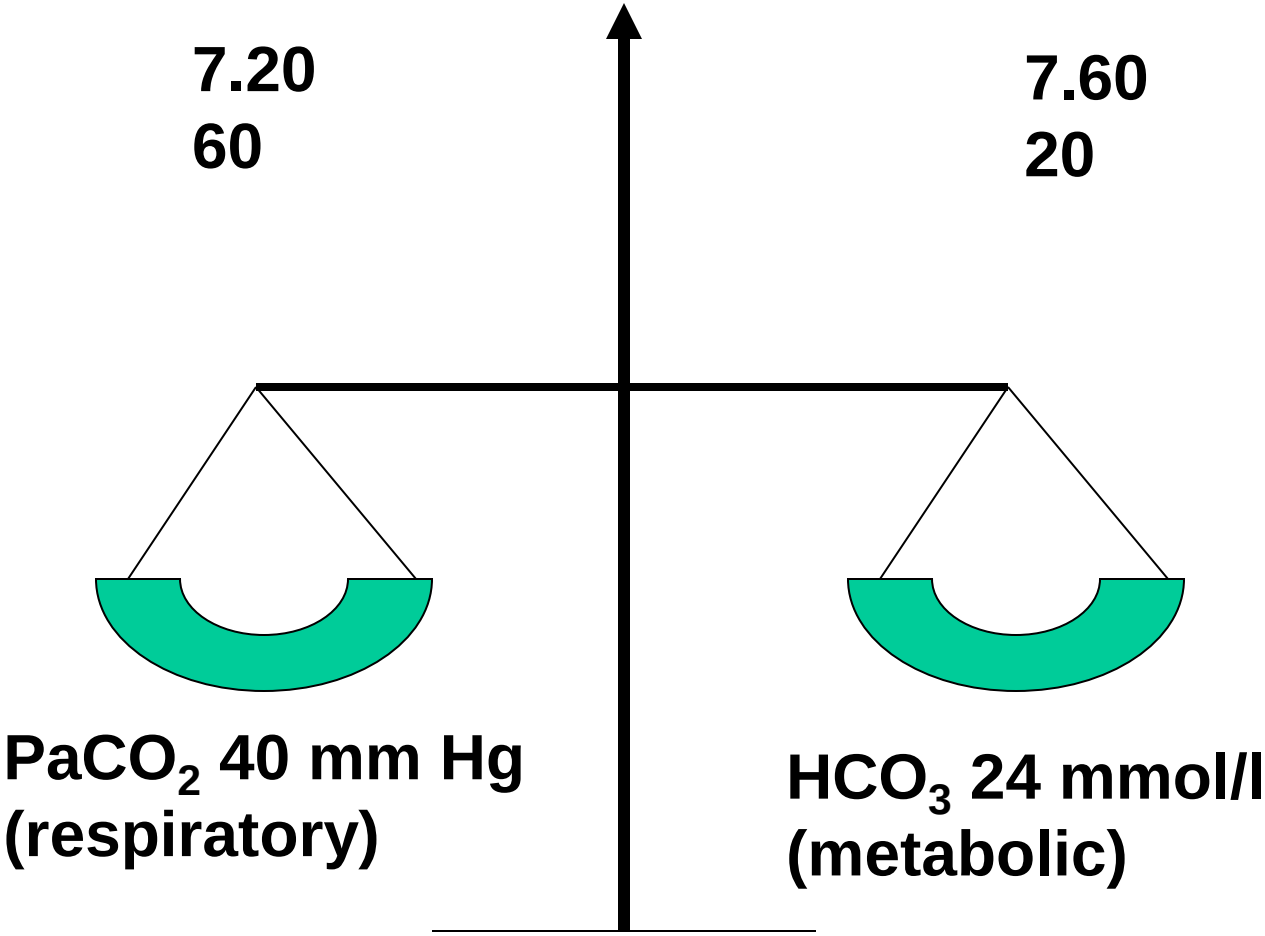


Questions to ask when patients have metabolic alkalosis

- **Is the ECV contracted?**
- **If the ECV is contracted, why?**
- **If the ECV is contracted, what is the renal response? The urine should be chloride free. If not, suspect diuretics**
- **If the ECV is <u>not</u> contracted, look for high aldosterone levels, high renin states, or primary hyperaldosteronism**

Acid-base „balance“

pH 7.4
[H] 40



Acid-base „balance“

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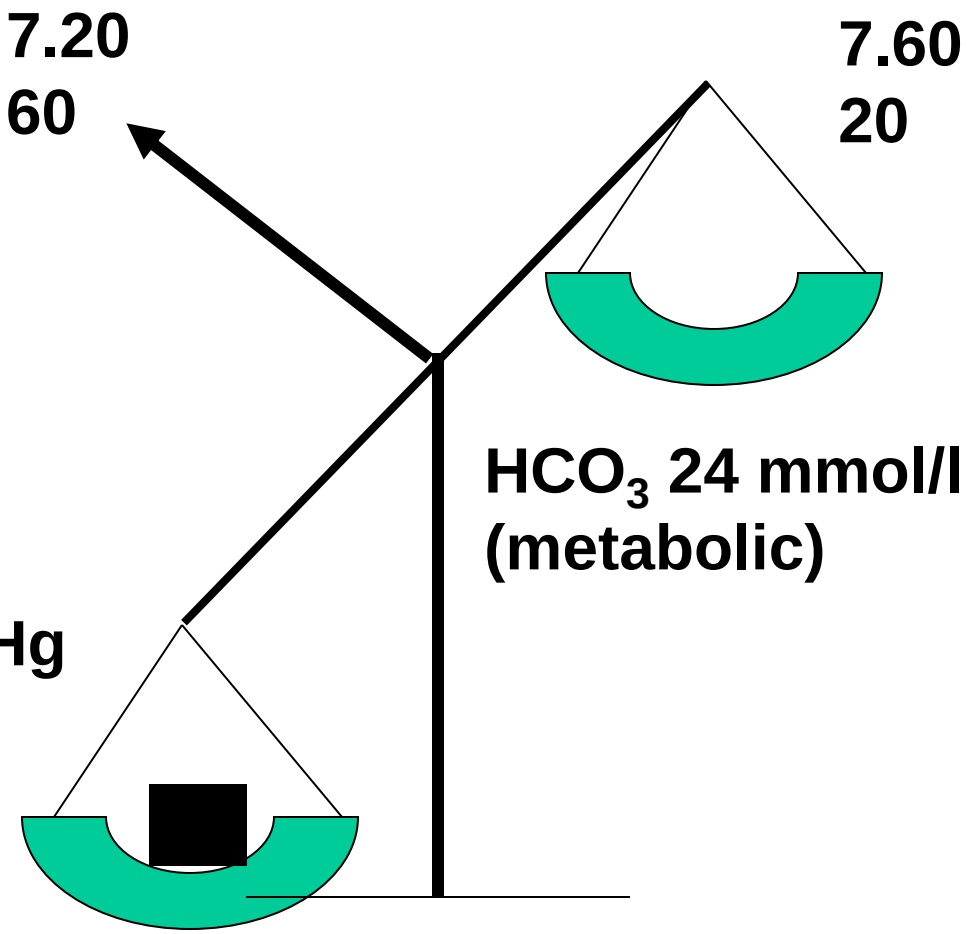
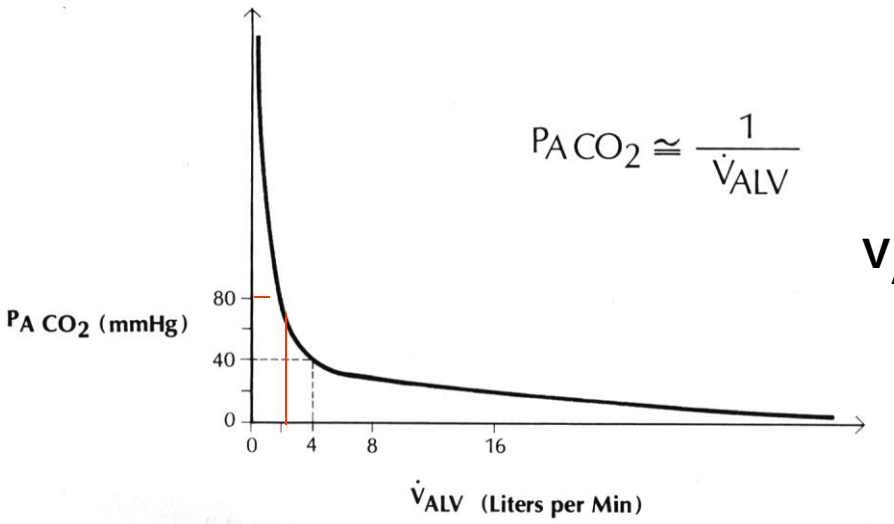


The respiratory component (H_2CO_3) is controlled by the lungs; we measure it as $PaCO_2$

The lungs excrete 20 000 mmol of CO_2 per day

The CO_2 content of a 70 kg person is 110 L

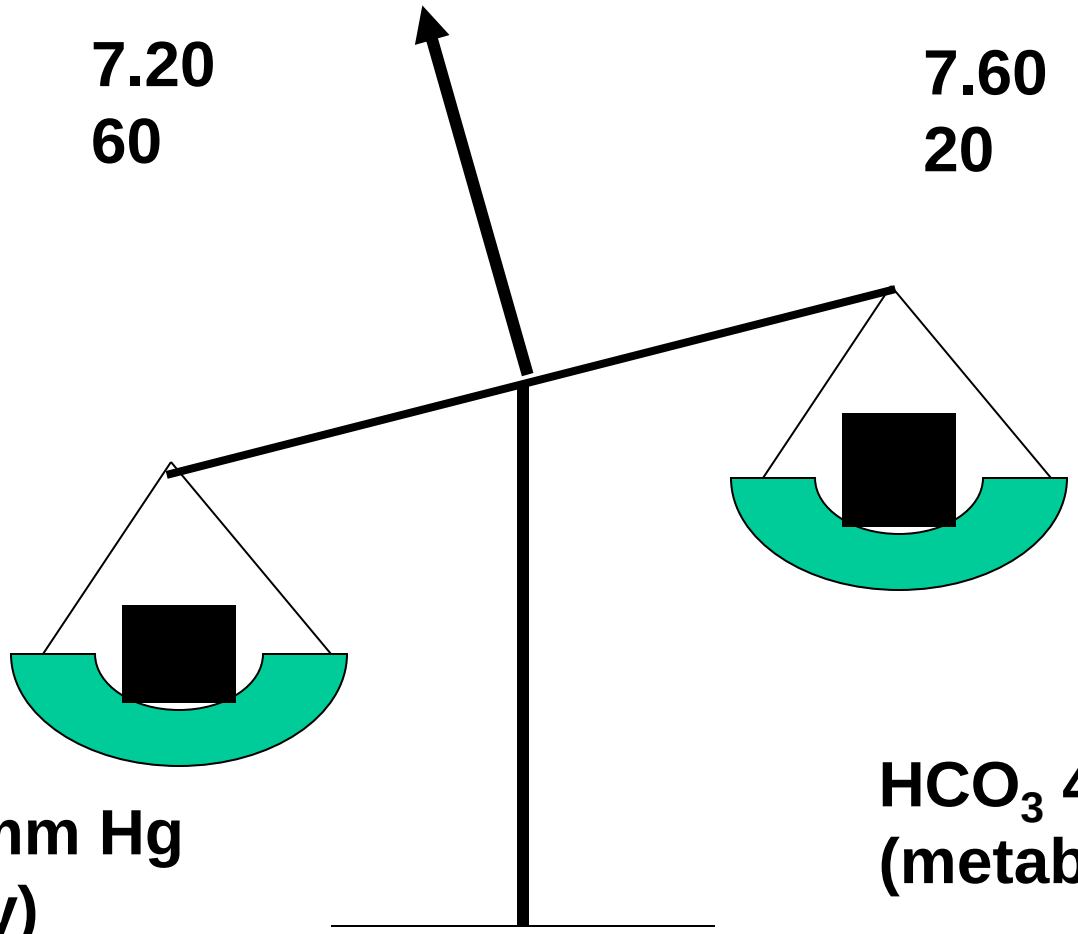
The $PaCO_2$ reflects the alveolar ventilation



$$V_{ALV} \sim \frac{gCO_2 \times V}{\bar{P}CO_2}$$

Acid-base „balance“

pH 7.4
[H] 40



$PaCO_2$ 89 mm Hg (respiratory)

HCO_3 47 mmol/l (metabolic)

50 year-old, obese, snoring, smoker

Measured values

- pH 7.329
- pCO_2 89.8 mm Hg
- pO_2 32.4 mm Hg
- SO_2 52.5%
- Hct 54%
- Hb 18,8 mg/dl
- Na^+ 138 mmol/l
- K^+ 4.17 mmol/l
- Cl^- 84.6 mmol/l
- Glu 7.2 mmol/l

Calculated values

- BEecf 21.5 mmol/l
- BE 14.5 mmol/l
- SBC 36.9 mmol/l
- HCO_3^- 47.7 mmol/l
- TCO_2 50.4 mmol/l
- A 44.6 mm Hg
- $A\text{-}aDO_2$ 12.3 mm Hg
- a/A 0.7
- An.Gap 10.0 mmol/l
- P50 27.9 mm Hg
- O_2 Cap 24.2 ml/dl
- O_2Ct 12.8 ml/dl

Temperatur-abhängig

Respiratory acidosis (a $PaCO_2$ problem) can come about by decreasing alveolar ventilation

- CO_2 and therefore H_2CO_3 is retained by the body
- The compensatory response must be metabolic, namely an increase in HCO_3

Acid-base balance summary

- Understand H^+ concentration and pH
- Understand the H_2CO_3 and HCO_3 buffering system
- Understand the primary disturbance and the compensatory response (1:1, 1:0.7, or 1:0.3)
- Know what compensation is appropriate
- Understand the anion gap and gain of H^+
- Understand gain of HCO_3 through loss of HCl or increased Na vs. H exchange
- Know alveolar ventilation - gain and loss of $PaCO_2$