METABOLIC ALKALOSIS

Dr.asadpour.ak

Intensivist

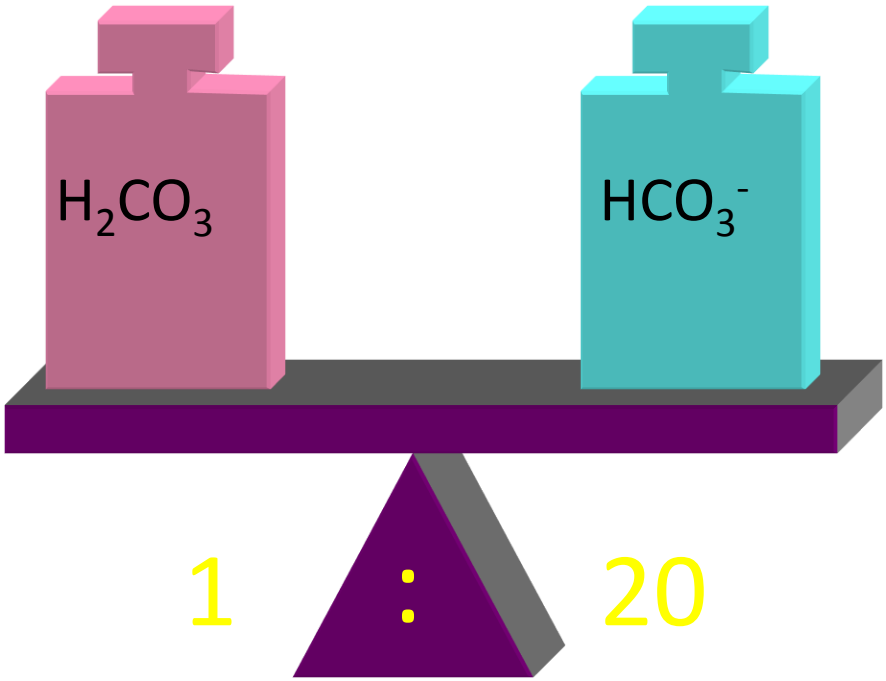
Shahrood Medical university

METABOLIC ALKALOSIS

- Elevation of **pH** due to an increased 20:1 ratio
  - May be caused by:
    - ***An increase of bicarbonate***
    - ***A decrease in hydrogen ions***
  - Imbalance again cannot be due to **CO_2**
  - Increase in **pH** which has a non-respiratory origin

7.4

METABOLIC ALKALOSIS



H_2CO_3 : Carbonic Acid

HCO_3^- : Bicarbonate Ion

$(Na^+)\ HCO_3^-$

$(K^+)\ HCO_3^-$

$(Mg^{++})\ HCO_3^-$

$(Ca^{++})\ HCO_3^-$

- **metabolic balance before onset of alkalosis**
- **pH = 7.4**

METABOLIC ALKALOSIS

- A reduction in **H^+** in the case of metabolic alkalosis can be caused by a deficiency of non-carbonic acids
- This is associated with an increase in **HCO_3^-**
- Can be the result of:

1) Ingestion of Alkaline Substances

2) Vomiting (loss of HCl)

METABOLIC ALKALOSIS

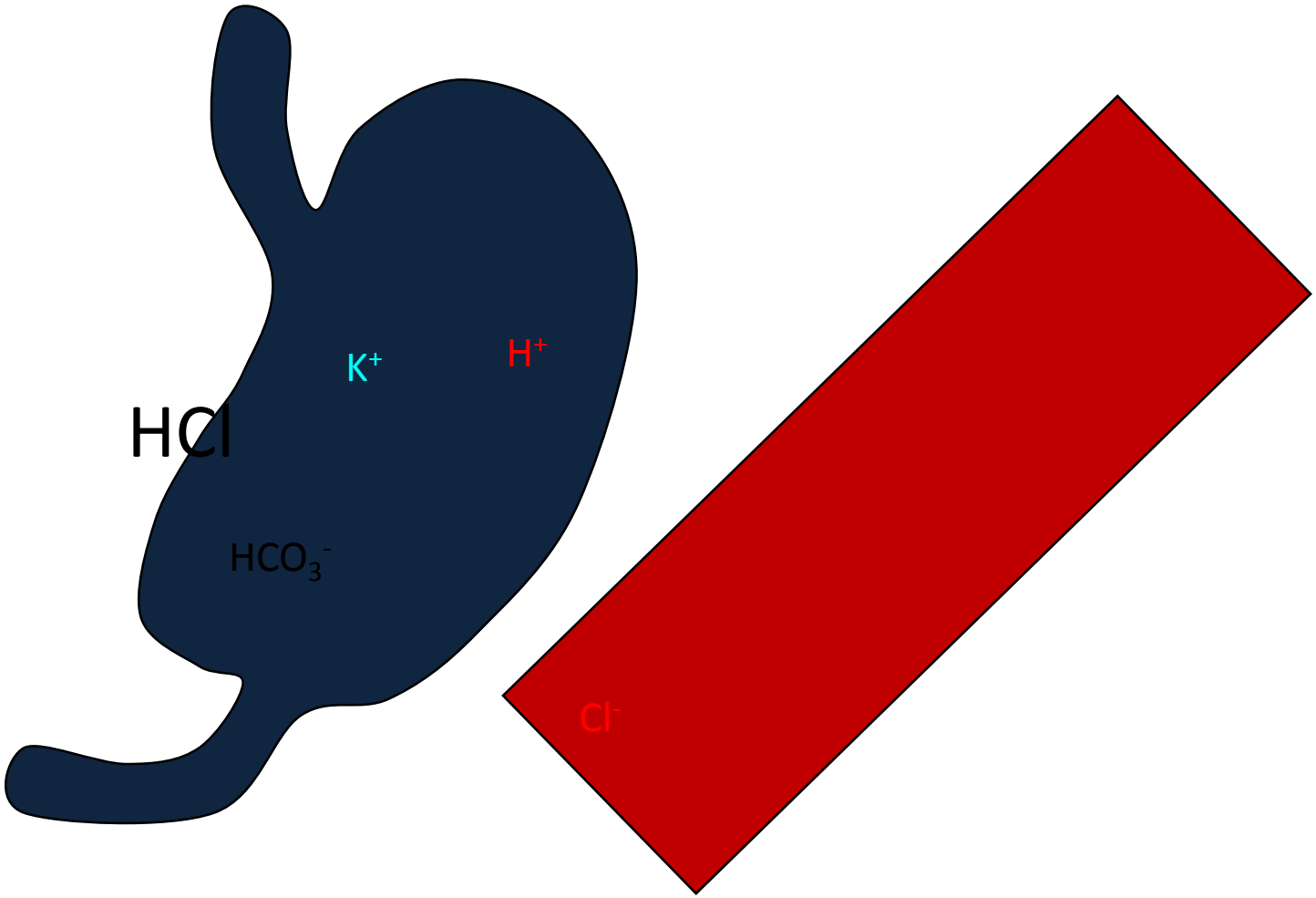
- ***1) Ingestion of Alkaline Substances***
  - Influx of **$NaHCO_3$**
    - Baking soda (**$NaHCO_3$**) often used as a remedy for gastric hyperacidity
      - **$NaHCO_3$** dissociates to **Na^+** and **HCO_3^-**
    - Bicarbonate neutralizes high acidity in stomach (heart burn)
    - The extra bicarbonate is absorbed into the plasma increasing **pH** of plasma as bicarbonate binds with free **H^+**

METABOLIC ALKALOSIS

- ***2) Vomiting (abnormal loss of HCl)***
  - Excessive loss of **H^+**

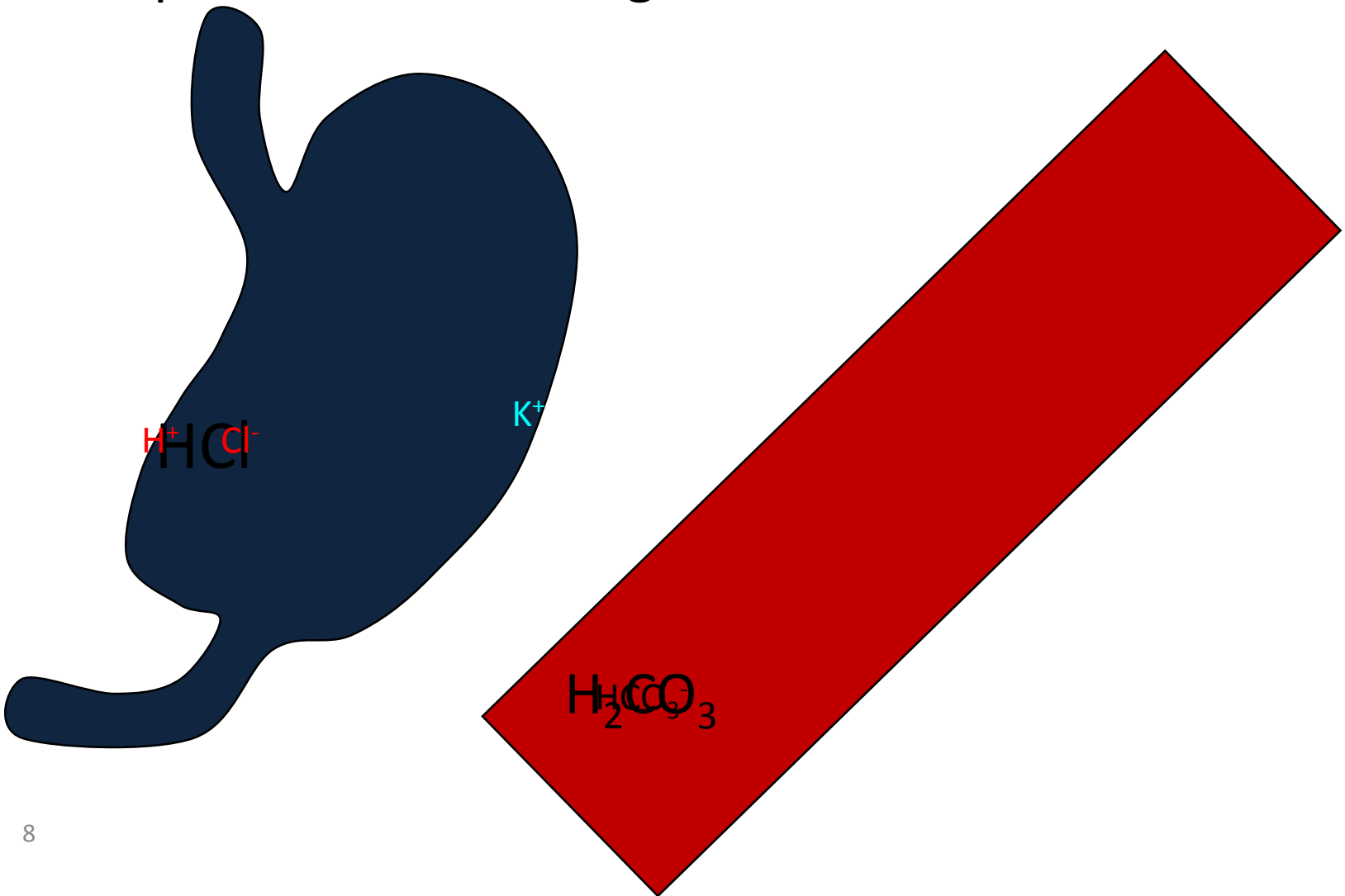
METABOLIC ALKALOSIS

- Gastric juices contain large amounts of **HCl**
- During **HCl** secretion, bicarbonate is added to the plasma



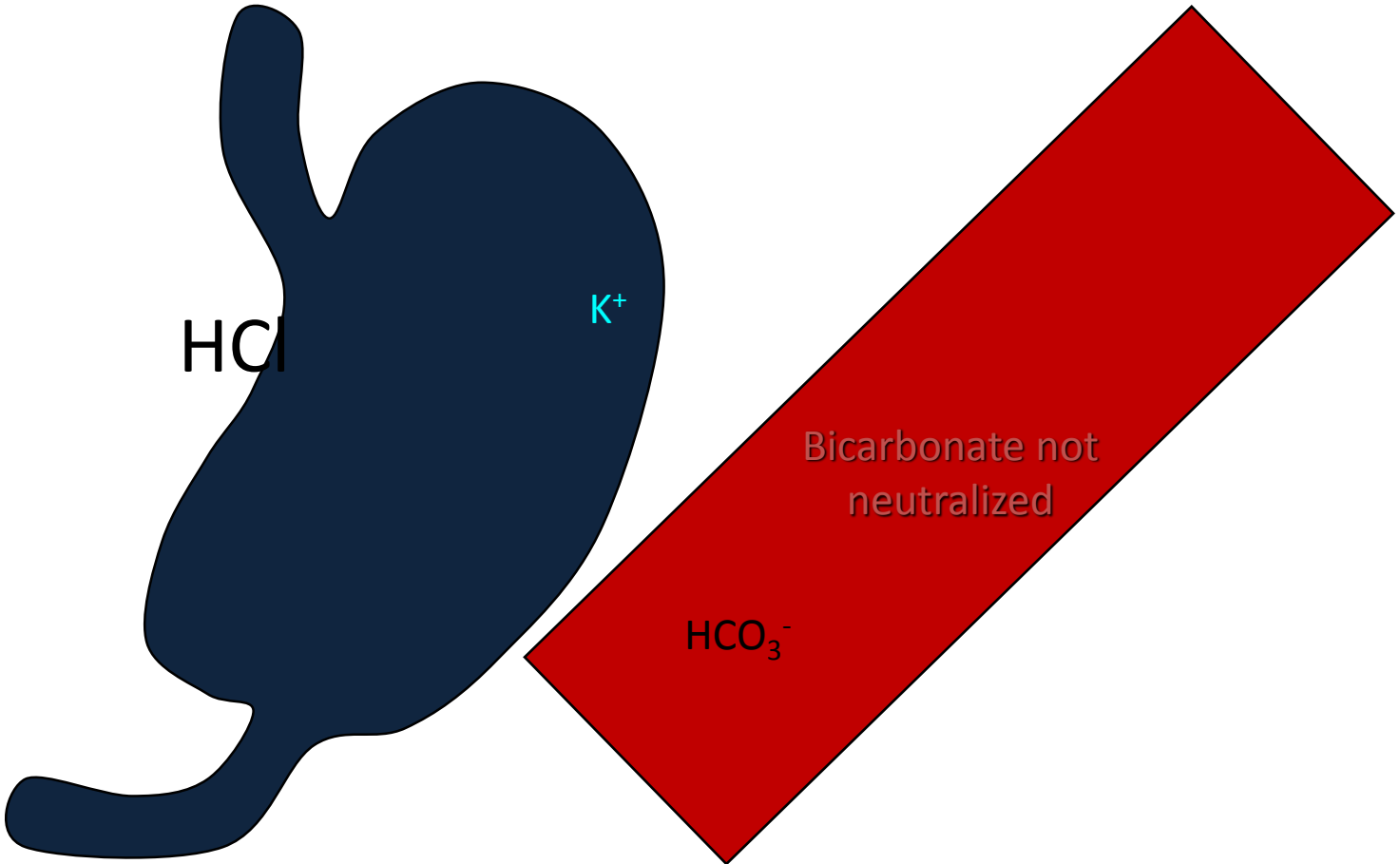
METABOLIC ALKALOSIS

- The bicarbonate is neutralized as **HCl** is reabsorbed by the plasma from the digestive tract



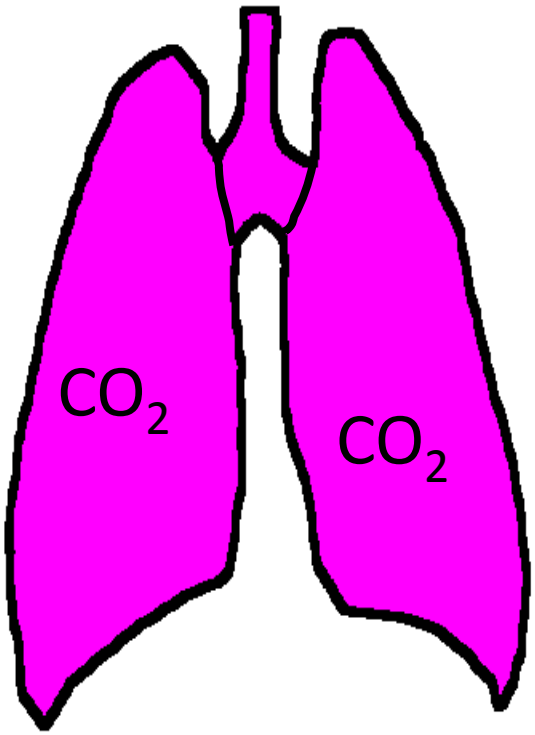
METABOLIC ALKALOSIS

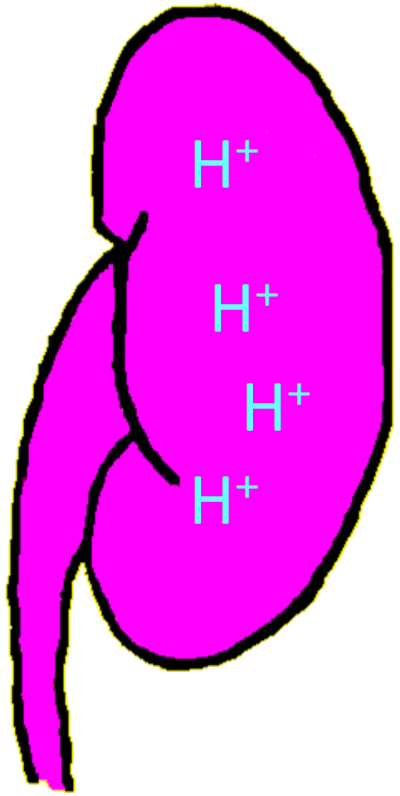
- During vomiting **H^+** is lost as **HCl** and the bicarbonate is not neutralized in the plasma
  - Loss of **HCl** increases the plasma bicarbonate and thus results in an increase in **pH** of the blood



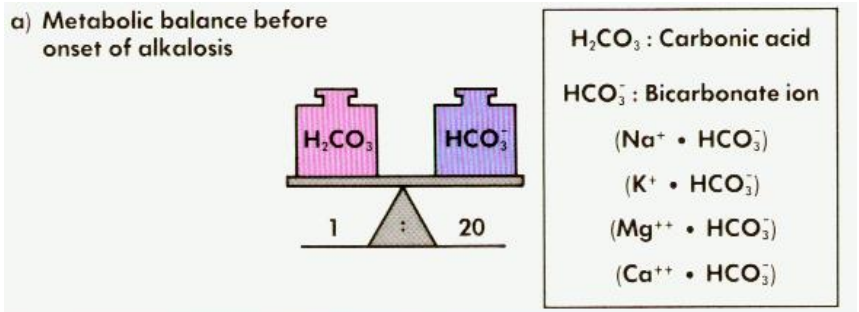
METABOLIC ALKALOSIS

- Reaction of the body to alkalosis is to lower **pH** by:
  - *Retain CO_2 by decreasing breathing rate*
  - *Kidneys increase the retention of H^+*

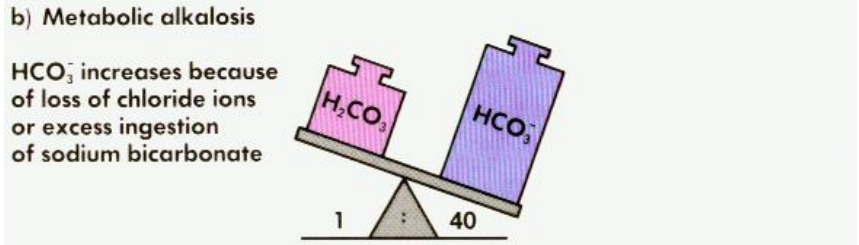




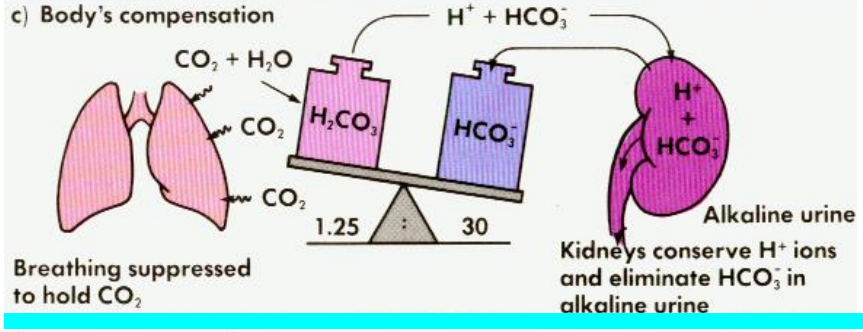
METABOLIC ALKALOSIS



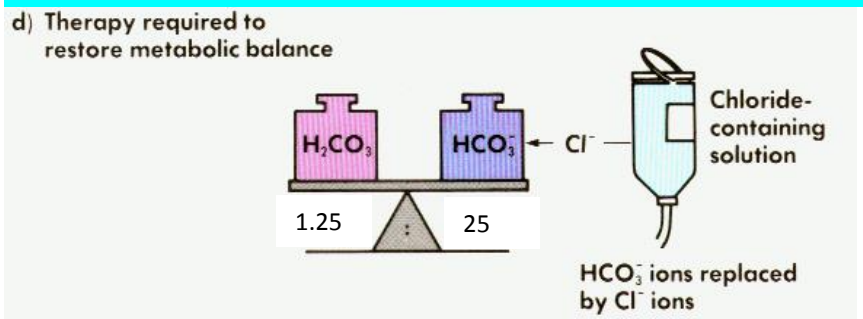
- metabolic balance before onset of alkalosis
- pH = 7.4



- metabolic alkalosis
- pH = 7.7
- HCO_3^- increases because of loss of chloride ions or excess ingestion of $NaHCO_3$

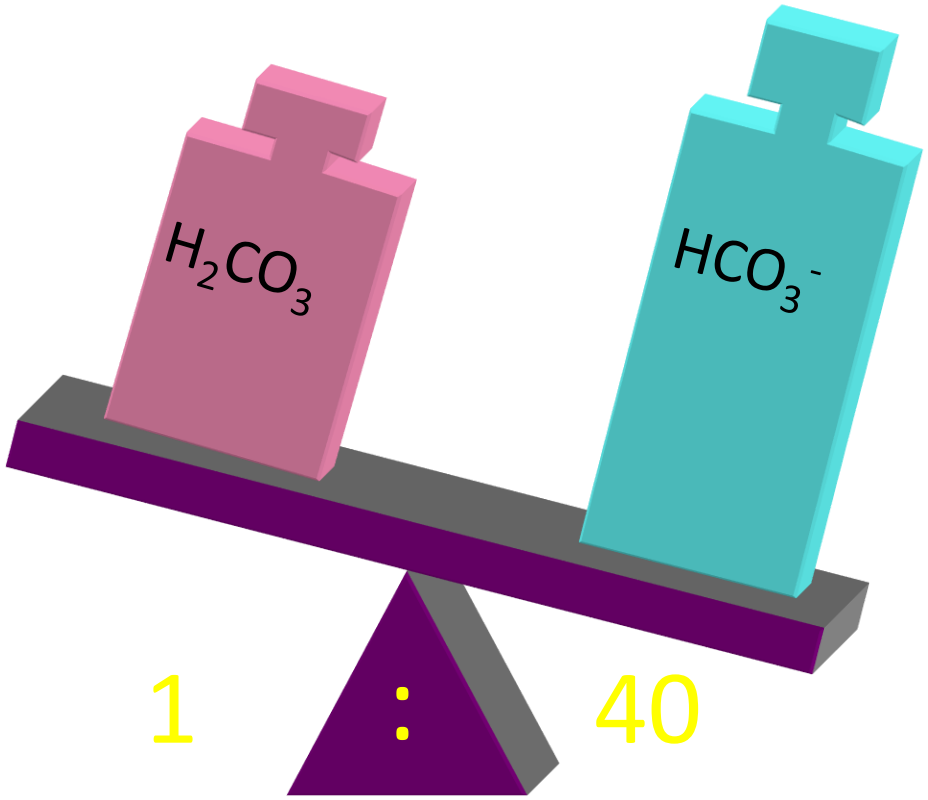


- body's compensation
- breathing suppressed to hold CO_2
- kidneys conserve H^+ ions and eliminate HCO_3^- in alkaline urine



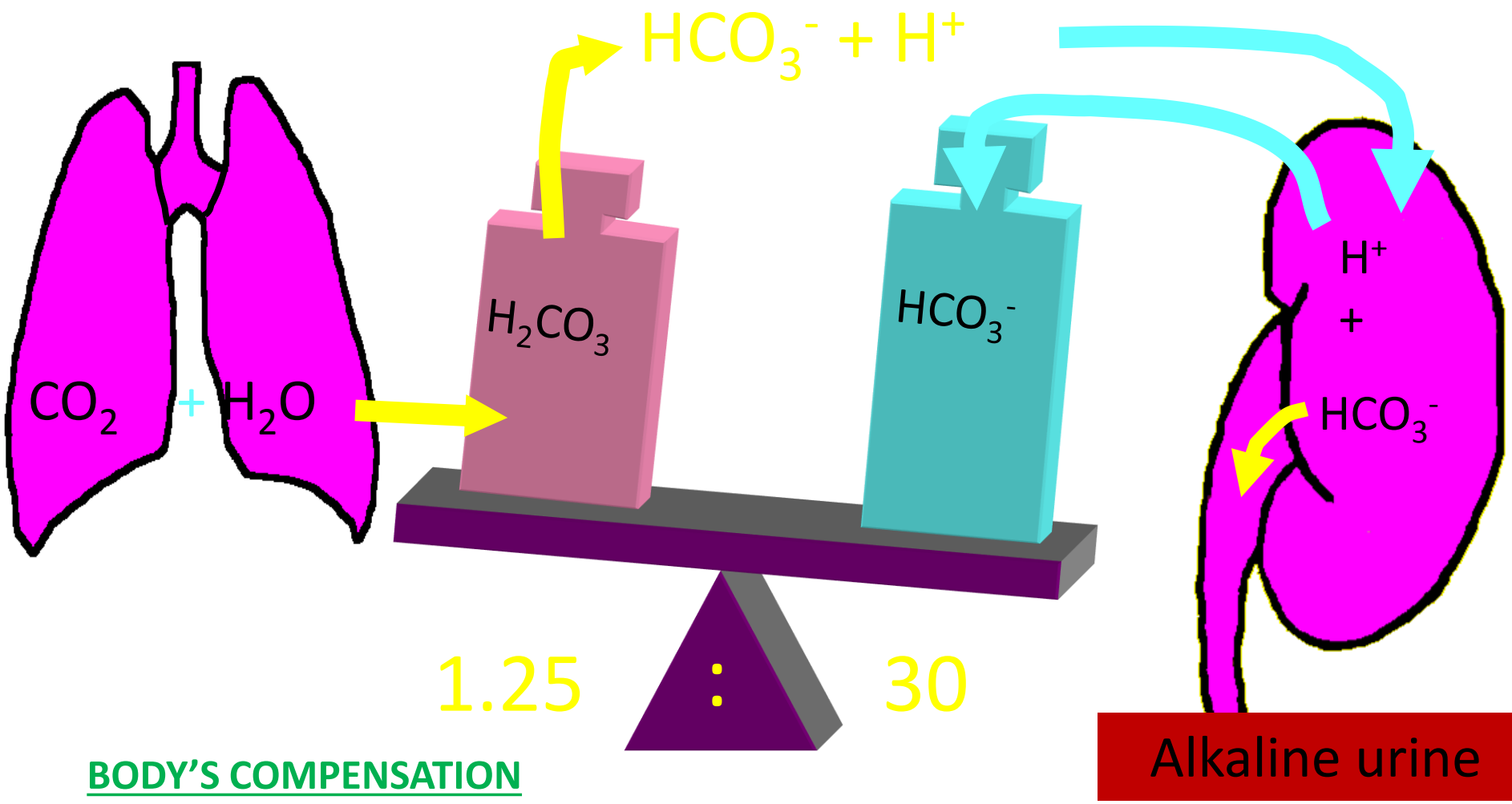
- therapy required to restore metabolic balance
- HCO_3^- ions replaced by Cl^- ions

METABOLIC ALKALOSIS



- **pH = 7.7**
- **HCO_3^- increases because of loss of chloride ions or excess ingestion of $NaHCO_3$**

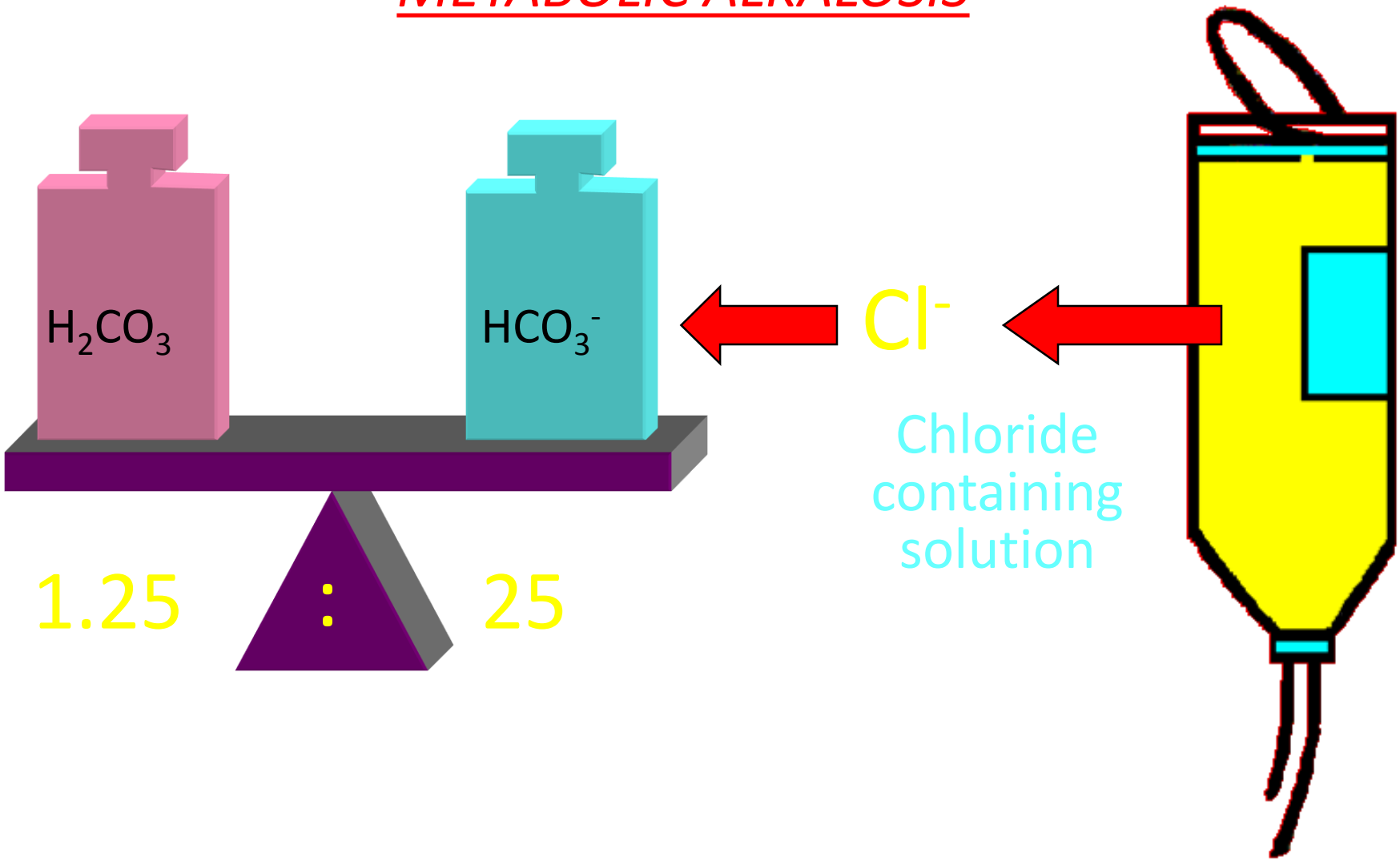
METABOLIC ALKALOSIS



BODY'S COMPENSATION

- **breathing suppressed to hold CO_2**
- **kidneys conserve H^+ ions and eliminate HCO_3^- in alkaline urine**

METABOLIC ALKALOSIS



- **Therapy required to restore metabolic balance**
- **HCO_3^- ions replaced by Cl^- ions**

Physiology

- Kidneys are responsible for maintaining a stable bicarbonate concentration
- Too much bicarb and it is rapidly excreted in the urine (eg. when you inject sodium bicarb into a healthy person, the pH only rises very briefly)
- Too little bicarb, and all of it gets reabsorbed from the urine

for alkalosis to persist, there needs to be an additional process which impairs renal bicarb regulation

How a metabolic alkalosis is initiated

- ***Gain of alkali***
  - ***From the outside*****,** eg. infusion of sodium bicarbonate
  - ***From the inside*****,** eg. metabolism of ketoanions to produce bicarbonate – like lactate in Hartmanns, acetate in Plasmalyte, citrate in transfused blood
- ***Loss of acid***
  - ***Through the kidneys*****,** eg. use of diuretic
  - ***Through the gut*** **,** eg. vomiting or NG suction

Causes of metabolic alkalosis

Chloride sensitive

Gastrointestinal

- **Vomiting**
- **Gastric drainage**
- **Chloride diarrhea**
- **Villous adenoma**

Renal

- **Diuretics**
- **Posthypercapnic**
- **Low chloride intake**

Sweat

- **Cystic fibrosis**

Causes of metabolic alkalosis

Chloride resistant

Increased minrealocorticoid activity

Primary hyperaldosteronism

secondary hyperaldosteronism

Cushing's syndrome

Licorice ingestion

Bartter's syndrome

Miscellaneous

Massive blood transfusion

Acetate-containing colloid solutions

Alkaline administration with renal insufficiency

Causes of metabolic alkalosis

Miscellaneous (cont)

- **Alkaline administration with renal insufficiency**
  - **Alkali therapy**
  - **Combined antacid & cation exchange resin therapy**
- **Hypercalcemia**
  - **Milk-alkali syndrome**
  - **Bone metastasis**
  - **Sodium penecillins**
- **Glucose feeding after starvatiion**

How a metabolic alkalosis is maintained

Whatever maintains the alkalosis has to cause a massive decrease in the kidneys ability to handle bicarbonate and hydrogen ions.

These causes can be divided into 4 groups:

- ***Chloride depletion***
- ***Potassium depletion***
- ***Reduced glomerular filtration rate***
- ***Extracellular volume depletion***

Chloride depletion alkalosis

- **The most common form: 90% of clinical cases**
- **Chloride and bicarb are the only anions present in any significant quantity in the ECF: decrease in one leads to an increase in the other**

Gastric acid loss eg. due to NG suction or vomiting:

Enteric chloride loss eg. due to villous adenoma

Diuretic use, eg. frusemide infusion

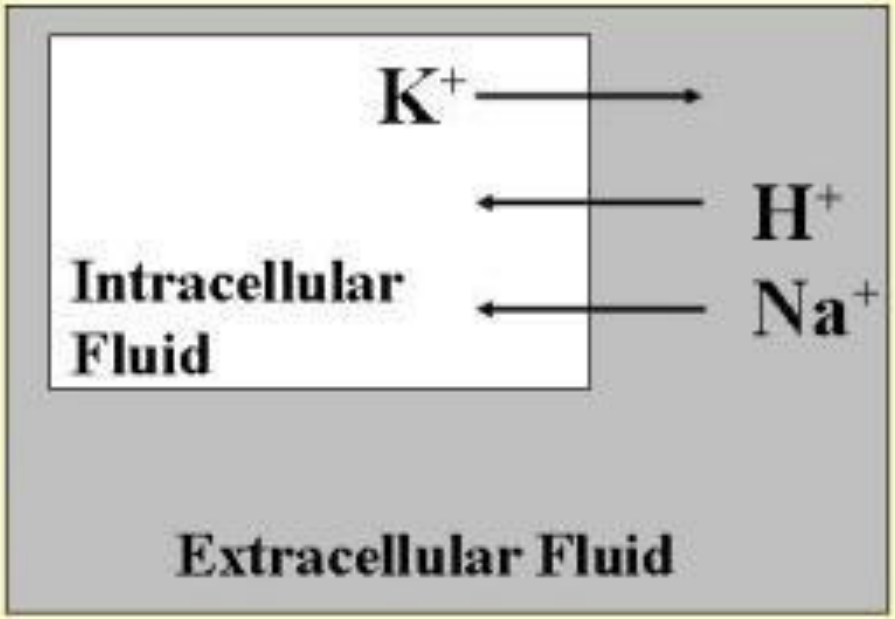
Administration of chloride is required to correct these disorders

Potassium depletion alkalosis

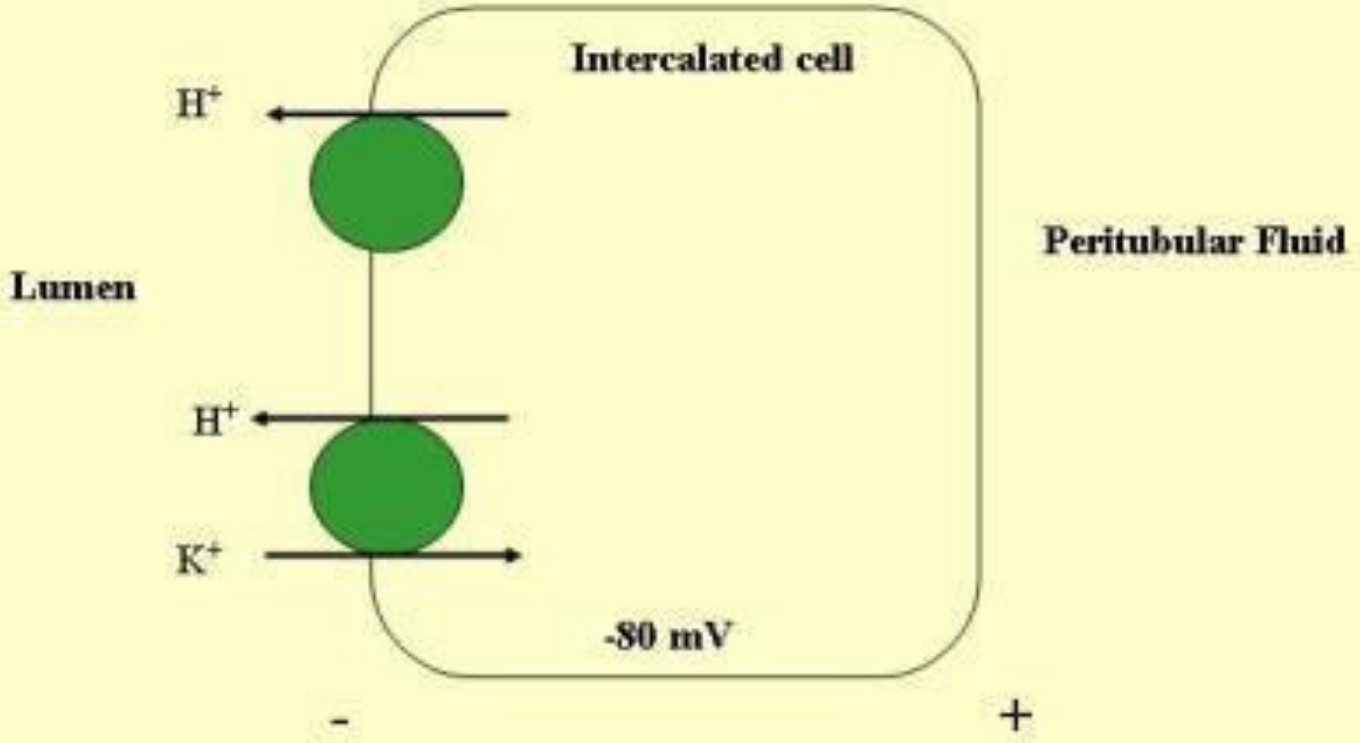
- **Bicarbonate resorption in the proximal and distal tubule is increased in the presence of potassium depletion**
- **Potassium depletion also decreases aldosterone release by the adrenal cortex**
- ***Examples:***
  - **Primary or secondary hyperaldosteronism** – increased aldosterone causes increased K+ and H+ losses
  - **Cushings syndrome, or COPD on IV hydrocortisone –** corticosteroids have some mineralocorticoid effect
  - **Potassium-depleting diuretics** eg. frusemide
  - Also **Bartter syndrome** (inherited juxtaglomerular hyperplasia), **licorice abuse** (pseudohyperaldosteronism)
  - **Severe potassium depletion alone**

Hypokalemia

- Metabolic alkalosis often causes K^+ depletion
- It is also exacerbated by K^+ depletion
- Extracellular K^+ shift is accompanied by intracellular H^+ and Na^+ movement
- H^+ movement increases serum HCO_3^- concentration
- Higher intracellular H^+ enhances H^+ secretion
- H^+-K^+ exchanger is up-regulated by K^+ depletion



H^+/K^+ Exchange Occurs in K+ Depletion



Another classification of metabolic alkalosis:

– Chloride responsive alkalosis - < 20mEq/L

- Loss of hydrogen ions, eg. vomiting
- Loss of bicarbonate-poor water, (concentration of bicarbonate, “contraction alkalosis”)

– Chloride resistant alkalosis - > 20mEq/L

- Retention of bicarbonate
- Shift of hydrogen ions into the intracellular space (eg. in hypokalemia)
- Administration of alkalotic agents, eg. bicarbonate

Mechanisms of Metabolic Alkalosis

A. Chloride responsive	B. Chloride resistant
Extracellular volume and chloride depletion	Direct stimulus to H^+ secretion
1. Vomiting 2. NG suction 3. Diuretics 4. Villous adenoma (5. Post-hypercapnia)	1. Primary aldosteronism 2. Cushing's syndrome 3. Steroid administration 4. Ectopic ACTH production 5. Adrenogenital syndrome 6. Licorice 7. Bartter's syndrome 8. Severe hypokalemia

Common causes of metabolic alkalosis in the INTENSIVE CARE UNIT

- Frusemide infusion, use of thiazides
- High volume NG aspirates
- Diarrhoea
- Severe hypokalemia (eg. insulin infusion)
- Corticosteroid therapy
- Overcorrection of chronic respiratory acidosis
- Recovery phase post organic acidosis
- Large doses of IV penicillin-based drugs

Consequences of metabolic alkalosis

- **In critically ill patients, = significant increase in morbidity and mortality**

- Decreased myocardial contractiity
- ➢ Arrhythmias
- Decreased cerebral blood flow (vasoconstriction)
- ➢ Neuromuscular excitability → tetany → difficult ventilation
- Impaired peripheral oxygen unloading
- ➢ Confusion, obtundation, seizures
- Hypoventilation, thus atelectasis
- ➢ Increased V/Q mismatch (alkalosis inhibits hypoxic pulmonary vasoconstriction)

Clinical features of metabolic alkalosis

- **Hypoventilation, even hypoxia**
- **Other changes are similar to those of hypercalcemia:**
  - **confusion, obtundation, seizures**
  - **paraesthesia**
  - **Muscle cramps, tetany**

Treatment of Metabolic Alkalosis

pH < 7.6, asymptomatic:

➡ ***volume depleted:*** give IV-NS and K+

➡ ***edematous but intravascularly depleted (CHF, nephrosis, cirrhosis):***

- give albumin to ↑ intravascular volume
- replete K+ aggressively,
- and consider Diamox

➡ ***volume expanded (rare)***: *give KCl po or IV*

Treatment of Metabolic Alkalosis

pH > 7.6, symptomatic:

NH4Cl or HCl IV

- 20-40 mEq/hr of 0.1 N or 0.15 N HCl through central line
- (NH4Cl must be avoided in cirrhosis or renal failure
- 100 mEq H+ should ↓ HCO3- by 7 mEq/L
- follow hourly pH, pCO2, HCO3-
- stop when pH < 7.6 and patient asymptomatiC

Acetazolamide (Diamox)

- watch for ↓ K+; must supplement with massive amounts KCl
- 250 mg IV Q8H
- stop when pH < 7.6 and patient asymptomatic

Treatment of Metabolic Alkalosis

Non-chloride-responsive:

Urinary Cl- > 20 mEq/l means not chloride-responsive (rare), so problem is probably primary hyperaldo, or severe lack of K+ leading to HCO3- loss;

give plenty of K+
give large doses of Aldactone (spironolactone)
maintenance therapy

METABOLIC ALKALOSIS

- Treatment of metabolic alkalosis is most often accomplished by replacing water and electrolytes (**sodium** and **potassium**) while treating the underlying cause

- Occasionally when metabolic alkalosis is very severe, dilute acid in the form of ammonium chloride is given by IV

Management of metabolic alkalosis in the Intensive Care Unit

Basic management:

- ***GIVE OXYGEN:*** the tissues are not getting enough
  - Unless the patient is a CO2 retainer with severe compensatory hypercapnea
- ***GIVE CHLORIDE:*** in chloride-responsive alkalosis this will reverse the alkalosis

"give chloride" means "give saline"

- ***GIVE PROTON PUMP INHIBITOR:*** if you reduce the rate of H+ excretion by the gastric mucosa

Management of metabolic alkalosis in the Intensive Care Unit

- ***REPLACE POTASSIUM / OTHER ELECTROLYTES***
- ***AVOID HYPERVENTILATION***
- ***STOP*** : NGT SUCTIONING,ANTIACID AND DIURETIC THERAPY

Management of metabolic alkalosis in the Intensive Care Unit

Advanced management strategies:

- ***Hydrochloric acid infusion***
  - Via a central line; just make sure it doesn't extravasate
  - The H+ will consume HCO3 – then its all about blowing off enough of the created CO2

- ***Acetazolamide***
  - Carbonic anhydrase inhibitor: forces kidneys to excrete HCO3 and H+ to enter the bloodstream together with CL-
  - Increases losses of Na+, K+, and water.