**Metabolic Alkalosis**

The persistence of a Met Alkalosis requires a process that acts to impair HCO3 excretion.

Metabolic effects:

1. decreased myocardial contractility
2. arrhythmias
3. decreased cerebral blood flow
4. confusion
5. mental obtundation
6. neuromuscular excitability

 impaired peripheral oxygen unloading (due shift of oxygen dissociation curve to left).

**Causes of a Met Alkalosis: Initiating Process**

Gain of Alkali in the ECF

* From an exogenous source (NaHCO3 infusion, citrate in blood)
* From an endogenous source (metabolism of keto-anions)

Loss of H+ from ECF

* Via kidneys (diuretics)
* Via Gut (vomiting, NG suction)

**Maintenance of the Alkalosis**:

Requires a process that impairs the kidneys ability to excrete HCO3 and prevent the return of the elevated plasma level to normal.

**Two Groups**

**1. Chloride depletion**: Cl- depletion causes the kidney to resorb more HCO3 than usual to maintain electro-neutrality as not enough Cl- around.

This is most common situation:

90% of Met Alkalosis is due to loss of gastric acid and diuretic use.

Vomiting due to pyloric stenosis or high obstruction will cause Met Alkalosis due to loss of HCl. Other causes of vomiting involve a mixture of acid and base loss from duodenum so Met Alkalosis less likely

Diuretics cause loss of chloride and Na in kidney so losses of Cl- exceed losses of HCO3. To become Alkalotic patients also have to be volume depleted and have decreases chloride intake (salt restricted). If enough Cl- in diet, unlikely to happen. Cl- in urine is only high after diuretic ingestion.

**2. K+ depletion**

Occurs in situation of mineralocorticoid excess, so low K+ leads to increased HCO3 resorption on prox and distal CT

Primary hyperaldosteronism: “saline resistant” Met Alkalosis. Increased Aldosterone levels lead to increased Na resorbtion and increased K+ and H+ loss. H+ loss is matched with increased HCO3 leaving the renal vein. Therefore metabolic alkalosis with low Cl-, K+ and Increased ECF volume.

Cushings: Excess steroids have mineralocorticoid effects.

Severe K+ depletion: Unknown cause of Met Alkalosis with low K+ and no excess mineralocorticoid activity.

Barters Syndrome: increased renin and aldosterone levels.

**Using Urine Chloride to differentiate between causes:**

Urine Cl- <10mmol/L

1. Volume depletion

2. Respond to saline infusion

3. Causes: Diuretics and Vomiting

Urine Cl- >20mmol/l

1. Volume expansion and hypokalaemia

2. Resistant to saline

3. Causes: excess aldosterone, severe hypoK, Barters, diuretic therapy (current, check urine later)

**Compensation**

Hypoventilation causes a rise in arterial CO2 but the magnitude and response is quite variable. May not occur due to

Hyperventilation due to pain, pulmonary congestion or due to hypoxia

**Expected pCO2 = 0.7xHCO3 + 20mmHg (range +/- 5mmHg)**

Wide range due to coexisting disorders as above.

A pCO2 well below this: Resp Alk also present.

**Tips for Assessment**

* High HCO3 and PCO2 suggest either Met Alk or Resp Acid
* If PCO2 > 60 either Met Alk very severe or Resp Acid present
* Suspect Met Alkalosis if Vomiting, NG suction, Pyloric obstruction, diuretic use or excess mineralocorticoid.
* Delta ratio useful to detect presence of a second met acid.
* If >2 either met acid present or rarely resp acidosis present
* Use a spot urine Cl-, if low probable Cl- depletion and need to replace
* If high, mineralocortcoid excess and need for K+ replacement
* Suspect surreptitious diuretic use in females, check UCl-