There are three different approaches to acid-base:

- Boston (approach below)
- Stewart (used by intensivists/anesthesiologists which looks at unusual parameters like SID)
- Copenhagen (used by surgeons/anesthesiologists which looks at base excess)

1. Look at pH
   - Acidemic (<7.35) vs Alkalemic (>7.45)
   - NB if pH is within normal limits this is not to say that everything is normal and that there are no acid-base disorders going on. There could be two simultaneous primary opposite disorders of different systems (metabolic and respiratory) or two metabolic disorders that cancel each other out. Hence look at (2)

2. Look at [HCO₃⁻] from venous blood BMP and pCO₂ from ABG to determine which one is causing the pH disturbance b/c one should reflect the pH and the other should reflect a compensation
   - [HCO₃⁻] = 22-26 mmol/L, Met Alkalosis: ↑ [HCO₃⁻] vs Met Acidosis: ↓ [HCO₃⁻]
   - pCO₂ = 35-45 mm Hg, Resp Alkalosis: ↑ pCO₂ vs Resp Acidosis: ↓ pCO₂
   - NB if both are in the same direction then two simultaneous primary same disorders (primary metabolic acidosis/alkalosis and primary respiratory acidosis/alkalosis) are occurring such that in the end there is a double disturbance
   - NB if both are w/in normal limits this is not to say that everything is normal and that there are no acid-base disorders going on rather there could be two simultaneous primary opposite disorders of the same system (metabolic only b/c you can’t have both resp acidosis and resp alkalosis) that cancel each other out

3. Calculate what the other parameter should be after compensation kicks in using the following equations:

4. Compare expected calculated compensation with the measured value
   - Normal Compensation (you have one primary problem with a normal compensation)
   - Over Compensation (you have another primary problem opposite to the first primary problem and in this scenario many times the pH is normal but [HCO₃⁻] and pCO₂ are not normal)
   - Under Compensation (you have another primary problem contributing to the first primary problem)
- Primary Metabolic Acidosis + Primary Respiratory Acidosis
- Primary Metabolic Alkalosis + Primary Respiratory Alkalosis

(5) Other useful calculations depending on disturbance
- (A) If MAc then calculate serum AG = [Na+] = ([Cl−] + [HCO3−]) = measured cations (MC) – measured anions (MA) ~ unmeasured anions (UA) – unmeasured cations (UC)
  - High AG (>12mEq/L) increase in an unmeasured anion (increase in endogenous (Lactate/Ketones/Other) or exogenous (Ingestants) organic non-Cl anion acids, hyperPO4, hyperalbuminemia) resulting in an increase in a measured cation (Na)
  - Decrease in an unmeasured cation (hypoCa, hypoMg, hypoK) resulting in a decrease in a measured anion (CI, HCO3−)
- Normal AG (~8mEq/L)
- Low AG (<4mEq/L) increase in an unmeasured cation (hyperCa, hyperMg, hyperK) resulting in an increase in a measured anion (CI, HCO3−)
  - Decrease in an unmeasured anion (decrease in endogenous (rare) organic non-Cl anion acids, hypoPO4, hypoalbuminemia therefore always correct AG for albumin: AG_corrected = AG + ¾[4 – albumin]) resulting in a decrease in a measured cation (Na)
- (B) If AGMAc then Calculate “delta+delta” = ΔAG + ΔHCO3− = determines if an additional metabolic acid-base disturbance exists (for every 1mEq of non-Cl acid added to circulation a 1mEq of HCO3− should be removed from circulation, if not 1:1 then some additional disturbance is accounting for the difference)
  - 0 = only an AGMA exists
  - + (less bicarb is being added) = AGMA + metabolic alkalosis
  - – (more bicarb is being removed) = AGMA + non-AGMA
- Other: Actual Base Excess Calculated (ABE_c) if you take a pt and make their pCO2 40mm Hg the amount of acid/base you have to add to make their pH 7.4 is there ABE:
  - – = add base
  - + = add acid

<table>
<thead>
<tr>
<th>Decreased Ventilation</th>
<th>Increased Ventilation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brainstem</td>
<td></td>
</tr>
<tr>
<td>Drug Induced (Opiates, Anesthetics, other sedatives)</td>
<td>Any Primary CNS Process</td>
</tr>
<tr>
<td>Oxygen administration in acute hypercapnia</td>
<td>Hypoxia</td>
</tr>
<tr>
<td>Central Sleep Apnea</td>
<td>Pain/Anxiety (catecholamines)</td>
</tr>
<tr>
<td>Lesions (Tumors, Infarcts, Trauma)</td>
<td>Sepsis (cytokines)</td>
</tr>
<tr>
<td>Metabolic (hypoTH)</td>
<td>Pregnancy (progesterone)</td>
</tr>
<tr>
<td>NM</td>
<td>Liver Disease (nitrogenous toxins like NH3)</td>
</tr>
<tr>
<td>Myopathy, NM Jxn Disease, Peripheral Nerve Dz, Radiculopathy/Plexopathy, Myelopathy (refer) esp Phrenic Nerve Paralysis from cardiac surgery, GBS, MG, damage to above C3-5 from trauma “3,4,5 keeps the diaphragm alive”, HypoPO4, HypoK</td>
<td>Medication (salicylates)</td>
</tr>
<tr>
<td>Pickwickian Syndrome (obesity induced hypoventilation)</td>
<td>Metabolic (hyperTH)</td>
</tr>
<tr>
<td>Lung</td>
<td></td>
</tr>
<tr>
<td>Parenchyma (pneumonia, pulm edema, restrictive lung dz, et al)</td>
<td>Pulmonary Embolus</td>
</tr>
<tr>
<td>Airway (obstructive lung dz, laryngobronchospasm, OSA, foreign body, et al)</td>
<td>Pneumonia</td>
</tr>
<tr>
<td>Pleura (pneumothorax, pleural effusion, fibrosis, et al)</td>
<td>Pulmonary Edema</td>
</tr>
<tr>
<td>Thoracic Cage (obesity, pregnancy, ascites,</td>
<td>Restrictive Lung Disease</td>
</tr>
<tr>
<td>Respiratory Acidoses</td>
<td>Respiratory Alkalosis</td>
</tr>
<tr>
<td>---------------------</td>
<td>-----------------------</td>
</tr>
<tr>
<td><strong>Etiology</strong></td>
<td>CNS (AMS, Paresthesia esp Perioral Numbness (2/2 cerebral vasoconstriction due to low CO₂))</td>
</tr>
<tr>
<td><strong>S/S</strong></td>
<td>CNS (AMS, Paresthesia esp Perioral Numbness (2/2 cerebral vasoconstriction due to low CO₂))</td>
</tr>
<tr>
<td>Where is the problem? Brainstem/NM/Lung (refer above)</td>
<td>CNS (AMS, Paresthesia esp Perioral Numbness (2/2 cerebral vasoconstriction due to low CO₂))</td>
</tr>
<tr>
<td>CV (Arrhythmias)</td>
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</tr>
<tr>
<td><strong>Tx</strong></td>
<td>Treat Underlying Cause</td>
</tr>
<tr>
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</tr>
<tr>
<td>Pulmonary Toilet</td>
<td>Breath into a Paper Bag which contains Increasing Amounts of CO₂ which decreases diffusion gradient for CO₂ from venous blood to air thus decreasing alkalosis (this doesn’t slow down your breathing just prevent too low CO₂)</td>
</tr>
<tr>
<td>Mechanical Ventilation (you want to decrease pCO₂ gradually b/c if rapid then neurologic problems including seizures/death)</td>
<td>Oxygen if hypoxic b/c can suppress resp drive</td>
</tr>
</tbody>
</table>

### Metabolic Acidoses

- **S/S**
  - CNS (AMS)
  - Pulm (Compensatory Hyperventilation w/ Kussmual Hyperventilation aka deep and fast)
  - CV (Arrhythmias/CHF/Vasodilation)
  - Metabolic (Bone Breakdown and Protein Catabolism)

- **Tx**
  - correct underlying cause and specific treatments below
  - MV if fatigued
  - RRT if severe
  - keep pH>7.2 and HCO₃<8 w/ acute IV NaHCO₃ or chronic PO NaHCO₃ (NB tastes bad and causes belching so some use KCitrate, Italian Briasci Soda 1drink TID, Baking Soda 1/2tsp TID)

- **Lactic Acid**
  - Type A (less oxidation of pyruvate and thus more conversion of pyruvate to lactate)
  - decreased oxygenation (cardiopulmonary failure w/ ischemia especially of bowel/limb, profound anemia, etc)
  - increased oxygen consumption (post-ictal, post-exertional, catechol excess, tumor, etc)
  - defect in oxygen utilization (1° salicylate, CO, CN, linezolid, NRTIs, tylenol, nitrates, etc)
  - Salicylates (early on there is a primary (seen usually in children) respiratory alkalosis 2/2 stimulation of CNS resp center followed by a primary (seen usually in adults) AG metabolic acidosis 2/2 lactic acid)
  - Type B (ni oxidation but there is failure of the liver to metabolize the small amount of lactate that is normally produced everyday 2/2 liver dz, DM, cancer, salicylates, alcohols, iron, isoniazide, inborn errors of metabolism)
  - D-LA (short bowel syndrome results in metabolism of glucose by colonic bacteria to D-lactate which is absorbed into blood but not measured by standard lactate assays b/c normally in the human body D-LA is never produced therefore if suspected then specifically order a D-Lactate)

- **Ingestant Acid** (Osmolar Gap (OG) = (measured osmoles) – (2Na + Glu/18 + BUN/2.8))
  - OG > 15 therefore Toxic Alcohol Ingestion (check “Volatile Acid Panel” in urine, formic/carboxylic/oxalic acid in serum, calcium oxalate in urine, fluorascene (component of antifreeze) in urine/clothes w/ woods lamp, Tx: (1) saturate/inhibit alcohol dehydrogenase w/ ETOH/Fomepizole, (3) Dialysis, (2) Folic Acid enhances metabolism of Formic Acid in Methanol vs Thiamine/Pyridoxine/Mg enhances metabolism of Oxalic Acid in Ethylene Glycol)
    - Methanol (aka Wood Alcohol, Windshield Wiper Fluid, Paint Thinner) → Formic Acid → ++ “drunk”, “snow storm” visual field changes, ab Sx (pain, N/V, pancreatitis)
    - Ethyl Alcohol (Regular Alcohol) → Aldehyde → Carboxylic Acid → ++ “drunk”
    - Ethylene Glycol (Antifreeze) → Oxalic Acid → +++ “drunk”, precipitates w/ calcium (calcium oxalate) in kidneys causing renal failure, in heart/lung causing cardiopulmonary failure, and in other vital organs, hypocalcemia
    - NB Isopropanol (Rubbing Alcohol) → Acetone (not an acid) → +++ “drunk”, gastritis, asphyxia (BUT NO AGMA, included here b/c one of the three toxic alcohols)
  - OG < 10 therefore Medication Overdose
    - Propylene Glycol found in many drugs that are given IV like benzos, dilantin, abx, etc (converted to Pyruvic Acid)
    - Acetaminophen (glutathione depletion resulting in accumulation of 5-Oxoproline)

- **Chronic Kidney Failure**
- **Keto Acid** (when there is low glucose in cells b/c of poor intake or low insulin, the liver converts FFAs into aceto-acetate (AcAc, measure in urine/plasma) and beta-hydroxy-butyrurate (B-OHB, cannot be measured) (NB AcAc + NADH ↔ B-OHB + NAD) where they are used as oxidative fuel by other organs esp heart/CNS)
- Diabetic KA (reduced cellular glucose intake) 3AcAc:1B-OHB
- Starvation KA (reduced oral glucose intake) 8AcAc:1B-OHB

Non-AGMAC

- GI Gain HCl/Equivalent (NH4-CI, Lysine-CI, Arginine-CI) where H then binds HCO3 and is converted into water therefore increase in CI and decrease in HCO3 hence nl AG
  - Ingesting/Injecting (TPN)
- GI Lose NaBicarb/Equivalent (Na-Lactate, Na-Citrate, Na-Acetate, Na-Butyrate) where there is an increase in Na and HCO3 hence nl AG
- Loss of Colon Contents (Diarrhea, Fistulas, High Output Ostomies, Uretero-Intestinal Diversion aka ileal Conduit (urine is diverted into the colon where it leads to increased GI reabsorption of chloride and GI secretion of bicarb))
- Impaired Kidney
  - Acute Kidney Failure (impaired acid filtering)
  - RTAs (impaired tubular function)

Other
- Post Chronic Hypocapnea (similar to hypocapnea below)
  - Expansion Acidosis aka Volume Expansion aka Dilutional (from rapid infusion of bicarb free IVF which has neutral pH of 7.0 hence in body it is “acidic”)
- NB UAG reflects Urine NH4 but very inaccurate

Renal Tubular Acidosis (RTAs) “241”

<table>
<thead>
<tr>
<th>Type II Proximal RTA</th>
<th>Type IV Distal RTA (most common)</th>
<th>Type I Very Distal RTA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Etiology For All: Idiopathic, Primary Congenital, s/p Renal Transplant, Tubulo-interstitial Dz</td>
<td>Defective ENaC at CD Principle Cells therefore decreased K+ secretion and thus +H+ excretion</td>
<td>Defective K+/H+ Antiporter at CD Alpha-Intercalated Cells therefore decreased H+ excretion</td>
</tr>
<tr>
<td>Defect</td>
<td>Moderate Acidosis</td>
<td>Severe Acidosis</td>
</tr>
<tr>
<td></td>
<td>Serum pH</td>
<td>Serum HCO3</td>
</tr>
<tr>
<td></td>
<td>Serum pH</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Urine pH</td>
<td>FeHCO3</td>
</tr>
<tr>
<td>Treatment</td>
<td>Easy to Tx just give HCO3/Aldo and decrease K</td>
<td>Easy to Tx just give HCO3 and replete K</td>
</tr>
</tbody>
</table>

NB Pt is usually asymptomatic until presenting w/
- Hypercalciuria (not sure if it is the cause or result of Type 1 RTA) and thus causing Nephrolithiasis and Medullary Calculifications aka Nephrocalcinosis
- Hearing Problems
- Cerebral Calculifications
- Osteopetrosis

Metabolic Alkalosis

- S/S (much less well tolerated than metabolic acidosis)
  - CNS (AMS)
  - Pulm (Compensatory Hypoventilation)
  - CV (Arrhythmias/CHF/Vasoconstriction)
Metabolic (hypoK/Ca/Mg/PO₄, shift in the oxyhemoglobin curve to the left such that Hgb is less willing to release oxygen to tissues, increased activity of intracellular enzymes requiring more ATP and thus more O₂)

- Tx
  - correct underlying cause and specific treatments below
  - MV if too much respiratory depression
  - RRT if severe
  - NS (if chloride sensitive) vs Aldactone/Diamox (if chloride resistant)
  - HCl

- Normally the kidneys have a large capacity to filter HCO₃⁻ therefore for metabolic alkalosis to occur two events must occur: (1) There must be increased HCO₃⁻ GENERATION and (2) the kidney must be impaired in its ability to handle the increased HCO₃⁻ generation resulting in a MAINTAINED metabolic alkalosis (normally causes metabolic acidosis b/c normally there are more acids than bases that are filtered... but if a pt has increased HCO₃⁻ production above in the presence of renal failure then metabolic alkalosis will occur)
  - GI Gain NaBicarb/Equivalent (Na-Lactate, Na-Citrate, Na-Acetate, Na-Butyrate)
    - Ingest/Injecting (baking soda, Milk-Alkali Syndrome (CaCO₃ supplements), IVF w/ bicarb, blood w/ citrate, TPN w/ acetate/glutamate, etc)
  - GI Loss HCl (NH₄-Cl, Lysine-Cl, Arginine-Cl)
    - Loss of Stomach Contents (when pts are actively gastric suctioned, vomiting, etc large amounts of acid are removed and in addition there is volume depletion, hypokalemia, and loss of chloride into stomach which stimulates bicarb reclamation), Tx: H₂B/PPI
  - Impaired Kidney
    - Opposite RTA-2 (Volume Contraction, etc results in proximal absorption of Na and thus loss of H and thus reclamation of HCO₃⁻)
    - Opposite RTA-4 (HyperAldo State, Increased Distal Delivery of Na as in chronic loop/thiazide diuretic use, etc)
    - Opposite RTA-1 (Hypokalemia, etc)
  - Other
    - Post Chronic Hypocapnea (after long standing resp acidosis the kidneys have time to compensate by retaining HCO₃⁻, when the resp disorder is acutely corrected as with mechanical ventilation the kidneys still retain HCO₃⁻ therefore you will have a transient alkalosis)
    - Contraction Alkalosis aka Volume Depletion (when a pt is on diuretics they are losing HCO₃⁻ poor fluid therefore when the blood “contracts” it contracts around a fixed amount of HCO₃⁻ resulting in an increase in [\[
\]] and in body that is considered “acidotic”, in addition proximal tubule will absorb Na to absorb more volume but in doing so HCO₃⁻ is absorbed and there is also stimulation of renin which leads to aldo and loss of H⁺ distally)

- NB diuretic use causes all of the “Opposite RTA” problems and “Contraction Alkalosis”
- NB GI Loss (volume contracted) VS GI Gain + Post Chronic Hypocapnea (non-volume contracted) hence assess volume status b/c some respond to fluids and others don’t and since fluids are so easy to give why not just start there, instead of looking at FeNa to estimate volume status (as you would in other situations) you shouldn’t in metabolic alkalosis b/c Na is altered and pulled into urine by HCO₃⁻ spill over, hence you can look at Urine [Cl] or even FeUrea
  - Low Urine [Cl] <20mEq/L = volume contracted states = responds to fluids = Saline Responsive
  - High Urine [Cl] >20mEq/L = non-volume contracted states = does not respond to fluids = Saline Resistant