Acid/Base						
Simple Acid Base Disorders						
Disorder	рН	pCO <sub>2</sub>	HCO₃			
Metabolic Acidosis	< 7.35	> 45	< 22			
Metabolic Alkalosis	> 7.45	< 35	> 26			
Respiratory Acidosis	< 7.35	> 45	< 22			
Respiratory Alkalosis	> 7.45	< 35	> 26			

- **Bold** indicates primary disturbance -- non-bold indicates secondary response.

  \*\*\*Lower serum bicarbonate levels (as low as 18 mmol/L) can be physiologically normal in neonates\*\*\*
  - Acidemia → pH < 7.35. Acidosis → process that makes pH ↓

<ul> <li>Acidemia → pH &lt; 7.35. Acidosis → process that makes pH ↑</li> <li>Alkalemia → pH &gt; 7.45. Alkalosis → process that makes pH ↑</li> <li>In respiratory disorders, the pH moves in the same direction as the pCO₂</li> <li>Always look at the pH! A high bicarb on a chem often represents a metabolic alkalosis, but could also be a compensation for chronic respiratory acidosis (e.g., in patients with chronic lung disease).</li> </ul>						
Metabolic Acidosis						
PowerPlans	M	Metabolism Lactic or Metabolic Acidosis NOS Admit Plan				
Approach	Us	Is there a concomitant resp acidosis / resp alkalosis? Use Winter's Formula Expected pCO2 = ([1.5 x HCO3-]+ 8 $\pm$ 2), then calculate AG $\rightarrow$ [Na+ – (Cl- + HCO3)]. Normal = 3*albumin +/- 2 (12 in healthy pts).				
Normal AG MAc		GI loss (diarrhea, laxative, ureteroenteric fistula) vs renal loss (RTA (see chart), acetazolamide use, renal failure (may also have elevated AG), aggressive rehydration with NS  • Can calc urine AG, (UNa + UK) – (UCI); if positive → impaired renal acidification, if negative → GI loss of bicarb, works b/c urine CI- = proxy for NH4+ secretion				
Renal Tubular Acidosis: Hyperchloremic Metabolic Acidosis w/ +Urine AG						
		Proximal (Type 2)	Distal (Type 1)	Hyperkalemic (Type 4)		
Defect		Bicarb Reabsorption	H+ secretion	Inadequate aldosterone		
Potassium		Normal/Decreased	Normal/Decreased	Increased		
Urine pH		< 5.5	> 5.5	< 5.5		
Renal stones		No	Yes (high urine pH → CaPhos stones, low urine citrate)	No		
Clinical correlates		Fanconi syndrome (generalized prox tubular dysfunction → lose glucose, phos, AAs)	Hereditary channelopathies (may be a/w SNHL)	DM, primary adrenal insufficiency, use of ACEIs/ aldo antagonists		
Increased AG MAc	MUDPILES Methanol Uremia Diabetic ketoacidosis/starvation ketoacidosis Paraldehyde Infection/Isoniazid/Iron/IEM Lactic Acidosis Ethylene Glycol Salicylates (cause primary metabolic acidosis and respiratory alkalosis)					