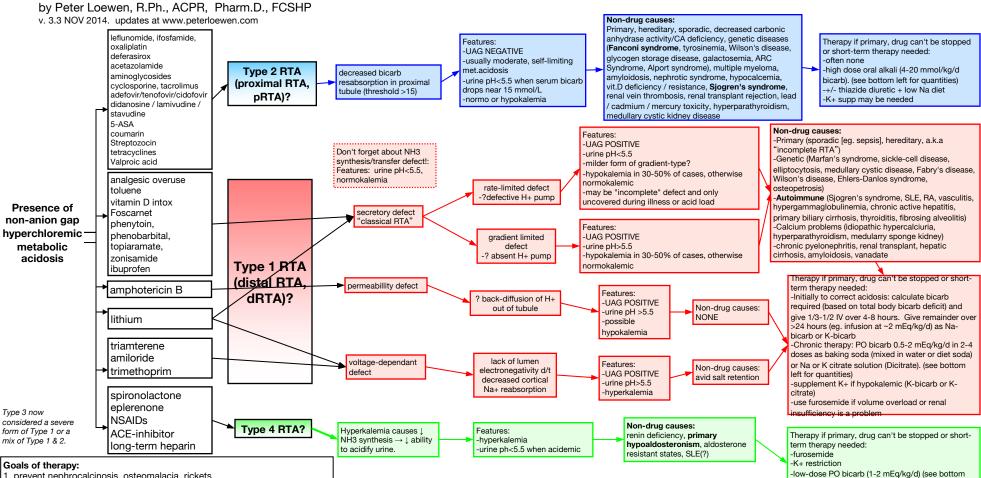
Renal Tubular Acidosis: A Drug-Centric Perspective



1. prevent nephrocalcinosis, osteomalacia, rickets

2. prevent severe acidosis, respiratory failure & death

3. prevent hypokalemic paralysis (with rhabdomyolysis), hyperkalemia & death

4. prevent irreversible renal damage (if drug-induced)

5. prevent fatique

NOTES:

-> UAG = urine (Na + K) - urine Cl

-> urine pH must be measured promptly (it increases as it sits since CO2 evaporates & bacteria produce urease -> increased urea... can't run pH on previously obtained sample)

-> most labs measure pH in 0.5 increments and only if >5.5

-> urine CI- is hard to do (not all labs can do it)

Alkali Replacement:

-> Total body bicarb deficit (mEq)= (desired - actual) x 0.6 x wt

-> Baking soda contains 12 mEq bicarb/g and ~270 mg Na+/g

-> K-citrate & Na-citrate (Dicitrate) contain 1mEq bicarb/mL (+ 1mEq K+ or Na+ per mL)

->NaBicarb tablets contain 325mg NaBicarb (=4 mEg bicarb + 90 mg Na+/tablet)

RENAL TUBULAR ACIDOSIS COMPARISON TABLE

Туре	Metabolic acidosis?	Anion gap	Serum CI-†	Serum K ^{+*}	UAG**	Urine pH
pRTA (Type 2)	\checkmark	Normal	\uparrow	Normal	NEG	<5.5 when acidemic
dRTA (Type 1)						
-NH ₃ synth / transport defect	\checkmark	Normal	\uparrow	Normal	POS	<5.5
-secretory defect	\checkmark	Normal	\uparrow	\downarrow in 30-50% of cases	POS	>5.5 (if gradient-limited, <5.5 if rate-limited)
-permeability defect	✓	Normal	\uparrow	↓ usually	POS	>5.5
-voltage defect	\checkmark	Normal	\uparrow	\uparrow	POS	>5.5
Туре 4	✓	Normal	\uparrow	\uparrow	POS	<5.5 when acidemic

†currently thought to be due to overactive CI-/HCO3- exchange pump in distal tubule (in Type1) [Pediatr Nephrol 2006;21:206-211] *when corrected for acidosis (eg. pH Δ 0.1 ~ K+ Δ 0.6 mmol/L)

**UAG = urine (Na + K) - CI

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left for quantities)

-fludrocortisone 50-200 mcg/d