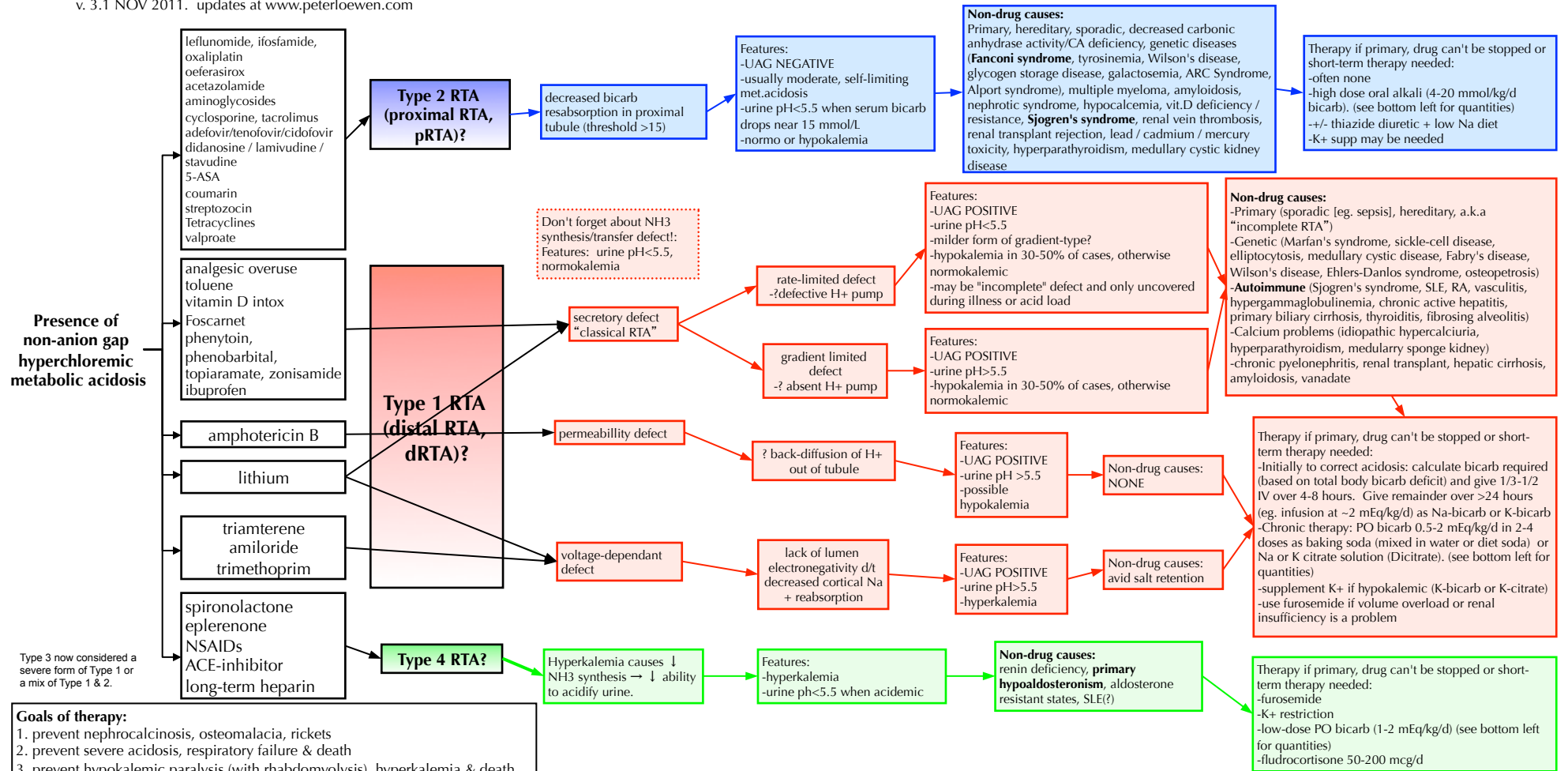


# Renal Tubular Acidosis: A Drug-Centric Perspective

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Type 3 now considered a severe form of Type 1 or a mix of Type 1 & 2.

## Goals of therapy:

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 fatigue

## 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 Cl- 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 mEq bicarb + 90 mg Na+/tablet)

## RENAL TUBULAR ACIDOSIS COMPARISON TABLE

Type	Metabolic acidosis?	Anion gap	Serum Cl <sup>+</sup>	Serum K <sup>**</sup>	UAG**	Urine pH
<b>pRTA (Type 2)</b>	✓	Normal	↑	Normal	NEG	<5.5 when acidemic
<b>dRTA (Type 1)</b>						
-NH <sub>3</sub> synth / transport defect	✓	Normal	↑	Normal	POS	<5.5
-secretory defect	✓	Normal	↑	↓ in 30-50% of cases	POS	>5.5 (if gradient-limited, <5.5 if rate-limited)
-permeability defect	✓	Normal	↑	↓ usually	POS	>5.5
-voltage defect	✓	Normal	↑	↑	POS	>5.5
<b>Type 4</b>	✓	Normal	↑	↑	POS	<5.5 when acidemic

†currently thought to be due to overactive Cl-/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) - Cl