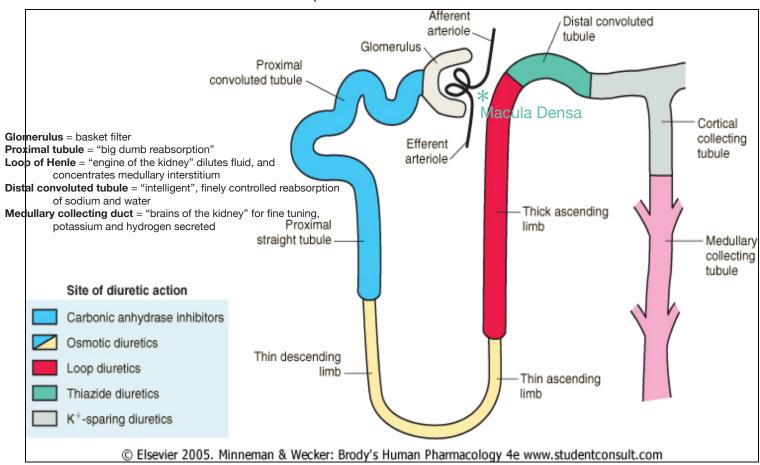
Nephron site of action of diuretics



From Glomerulus, closest to farthest

Carbonic anhydrase inhibitors and osmotic

acetazolamide (Diamox)

Loop Diuretics

furosemide (Lasix) "Lasts 6" (oral and IV) bumetanide (Bumex) torsemide (Demedex) Act immediately after glomerulus

Act on ascending Loop of Henle. Block Na/K/2Cl cotransporter, inhibit reabsorption into tissue. More effective in patients with impaired kidney function.

2° effect is increased prostaglandins, which results in vasodilation and increased blood supply to the kidney. NSAIDs block the COX pathway that synthesizes prostaglandins, so NSAIDs can reduce the efficacy of loop diuretics.

Thiazide Diuretics

hydrochlorothiazide (aka HCTZ) chlorthalidone metolazone (Zaroxolyn) (much more potent) Act on Distal Convoluted Tubule. Inhibit the sodium-chloride symporter. More effective in patients with normal kidney function. Used mostly for HTN, often with ACE-I, ARB, β-B, K+ sparing

See the 1st The Curbsiders episode with Joel Topf (also the reboot): • 30:18 Chlorthalidone versus hydrochlorothiazide

K+ Sparing Diuretics

triamterene (Dyrenium) amiloride (used mostly for HTN + CHF) Aldosterone Blockers - spironolactone (Aldactone) - eleprenone (Inspra) Aldosterone increases blood pressure through sodium retention and also decreasing potassium through excretion. Consider hyperaldosteronism as a cause of HTN in patients with low K+

Dyazide and Maxzide are (HCTZ(thiazide) plus triamterene (K+) combo)