

## ACE-I (Angiotensin Converting Enzyme Inhibitors)

benazapril (Lotensin) amlodipine/benzapril (Lotrel) benzapril/HCTZ (Lotensin-HCT)  
captopril (Capoten) captopril/HCTZ (Capozide)  
enalapril (Vasotec) enalapril/felodipine (Lexxel) enalapril/HCTZ (Vasarectic) enalaprilat (Vasotec IV)  
fosinopril (Monopril) fosinopril/HCTZ (Monopril-HCT)  
lisinopril (Prinivil, Zestril) HCTZ/lisinopril (Prinzide) HCTZ/lisinopril (Zestoretic)  
moexipril (Univasc) HCTZ/moexipril (Uniretic)  
perindopril (Aceon)  
quinapril (Accupril) HCTZ/quinapril (Accuretic)  
ramipril (Altace)  
trandolapril (Mavik) trandolapril/verapamil (Tarka)

- ramipril has the best data but in general all are similar therefore just the cheapest which is lisinopril
- all are PO and the only one that can be given IV is Vasotec
- 1/2 dose if elderly, concomitant diuretic use, and renal dz

## ARBs (Angiotensin II Receptor Type I Blockers)

candesartan (Atacand) candesartan/HCTZ (Atacand-HCT)  
eprosartan (Teveten) eprosartan/HCTZ (Teveten-HCT)  
irbesartan (Avapro) HCTZ/irbesartan (Avalide)  
olmesartan (Benicar) amlodipine/olmesartan (Azor) HCTZ/olmesartan (Benicar-HCT)  
losartan (Cozaar) HCTZ/losartan (Hyzaar)  
telmisartan (Micardis) telmisartan/HCTZ (Micardis-HCT)  
valsartan (Diovan) HCTZ/valsartan (Diovan-HCT) amlodipine/valsartan (Exforge)

- all are the same (class effect) therefore pick the one that is cheapest but unfortunately they are all expensive
- 1/2 dose if elderly, concomitant diuretic use, liver disease (losartan only), and renal dz
- There is evidence that ARBs have reduced incidence of new onset DM 2/2 stimulation of peroxisome proliferator activated receptor-gamma

## DRI (Direct Renin Inhibitors)

aliskiren (Tekturna)

Angiotensinogen (Liver)

↓ Renin (Kidney)

Angiotensin I = AI

↓ Angiotensin Converting Enzyme = ACE (Lung)

Angiotensin II = AII

### Mechanism

- ↓ AII formation
  - Decreased AII-Receptor Type I Activation
    - decreased vaso>venoconstriction → decreased afterload (rapid)
    - decreased proximal Na and thus water reabsorption → decreased preload (slow)
    - decreased Aldo release from adrenal cortex → decreased distal Na and thus water reabsorption → decreased preload (slow)
    - decreased glomerular efferent arteriole vasoconstriction (rapid)
    - decreased sympathetic tone (rapid)
    - decreased heart remodeling (slow)
    - decreased kidney remodeling (slow)
  - Decreased AII-Receptor Type II Activation (if present must switch to ARBs)
    - ↓ Bradykinin Degradation = ↑ Bradykinin (mast cell mediator)
    - ↓ Substance P Degradation = ↑ Substance P (vasodilator)

### Side Effects & Contraindications

#### CV

- hypotension
- neutropenia

#### Pulm

- chronic involuntary dry cough 2/2 increased Bradykinin / Substance P, seen in 12% of pts, w/in mos of initiation

#### GI

- change in taste (dysguesia)

#### Renal (monitor Cr and K+ @ 1wk after initiation or dose increase and then Q3-6mo)

- acute interstitial nephritis
- pre-renal ARF if bilateral renal stenosis, HF, hypovolemia, pre-existing RF
- hyperK

#### Skin

- dermatitis 2/2 increased Bradykinin / Substance P, seen in 10% of pts
- angioedema (soft tissue edema of the face, lips, tongue, oropharynx, epiglottis) 2/2 increased Bradykinin / Substance P, seen in 0.4% of pts, w/in 2wks of initiation (sometimes mos to yrs), check a C40 level, if important to use RAAS inhibitors then switch to ARB/DRI but tell pt risk still exists but most doctors don't use RAAS inhibitors at all

#### Ob/Gyn

- renal teratogenic



Copyright 2015 - Alexander Mantas MD PA