

# Renal A & Pee

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# **Time to refresh the memory with a little gross anatomy**

- Kidneys located in the retroperitoneal space between T12 and L3
- Right lower than the left

# The kidney...retroperitoneal space

- CVA tenderness
- Acute **pyelonephritis**
- **Glomerulonephritis**
- Palpation? Can you palpate the kidney in an adult?
- Not unless the kidney is HUGE...
- Polycystic kidney disease (PKD)

# Gross anatomy

- Renal cortex
- Renal medulla
- Renal pyramids
- Renal papillae
- Medullary interstitium
- Calyces
- “renal pelvis” — “pyels”

# Associated structures

- Plus the associated structures (ureters, bladder, urethra)
- Ureters and kidney stones
- Bladder and cystitis
- Urethra and estrogen

# The importance of estrogen and the maintenance of urinary tract health

- Estrogen receptors and the urethra
- Prepuberty , perimenopause, and postmenopause
- *E.Coli* and the rectum

# Gross Anatomy—blood supply

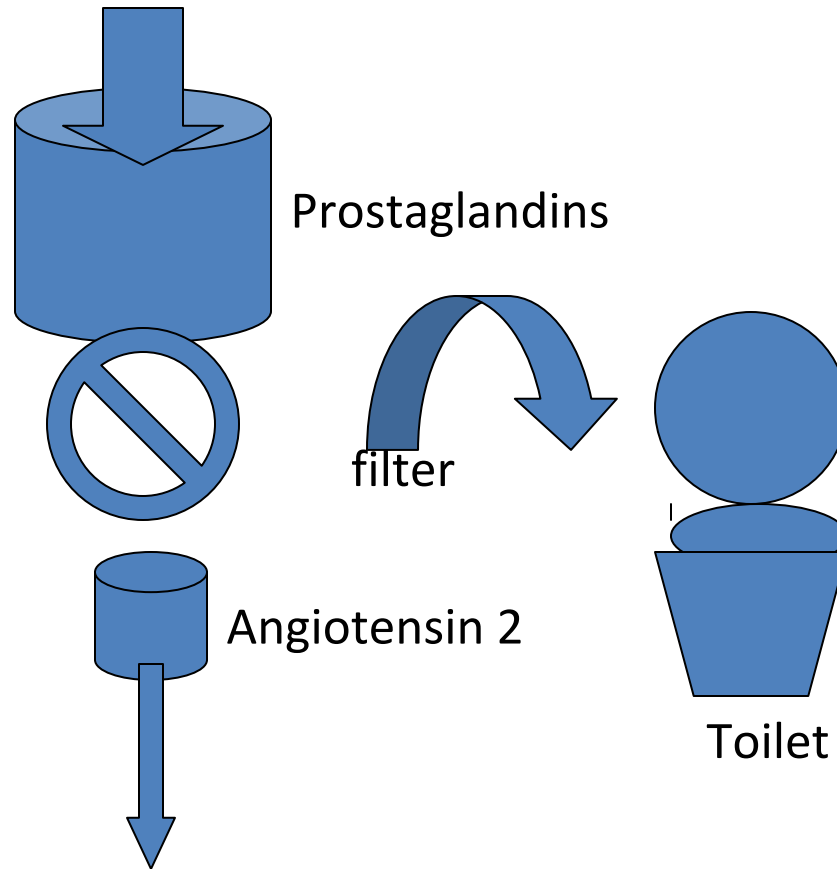
- Aorta→renal artery→branches into arcuate arteries→

# Blood supply to and from the glomerulus

- Afferent arteriole delivers blood to the
- Glomerulus—a tuft of capillaries
- Blood exits via the efferent arteriole

# Normal glomerular blood flow

- **Afferent arteriole**  
(vasodilated via  
(prostaglandins))
- **Blood entering**  
**glomerulus**
- **Glomerulus** → **filter**
- **Efferent arteriole**  
(vasoconstricted via  
(angiotensin 2))
- **Blood exiting**  
**glomerulus**



# What can go wrong with renal blood flow?

- Atherosclerosis of aorta and renal artery

In addition to decreasing blood flow to the kidney, what else does hyperlipidemia do?

- Hyperlipidemia is a disease-promoting factor thought to perpetuate glomerular injury and proliferation of the mesangial cells. Both epithelial cells and mesangial cells have receptors for LDL
- The “statin sisters” to the rescue

# Who are the “statin sisters”?

- Lovastatin (Mevacor)
- Pravastatin (Pravachol)
- Fluvastatin (Lescol)
- Rosuvastatin (Crestor)\*\*
- Atorvastatin (Lipitor)

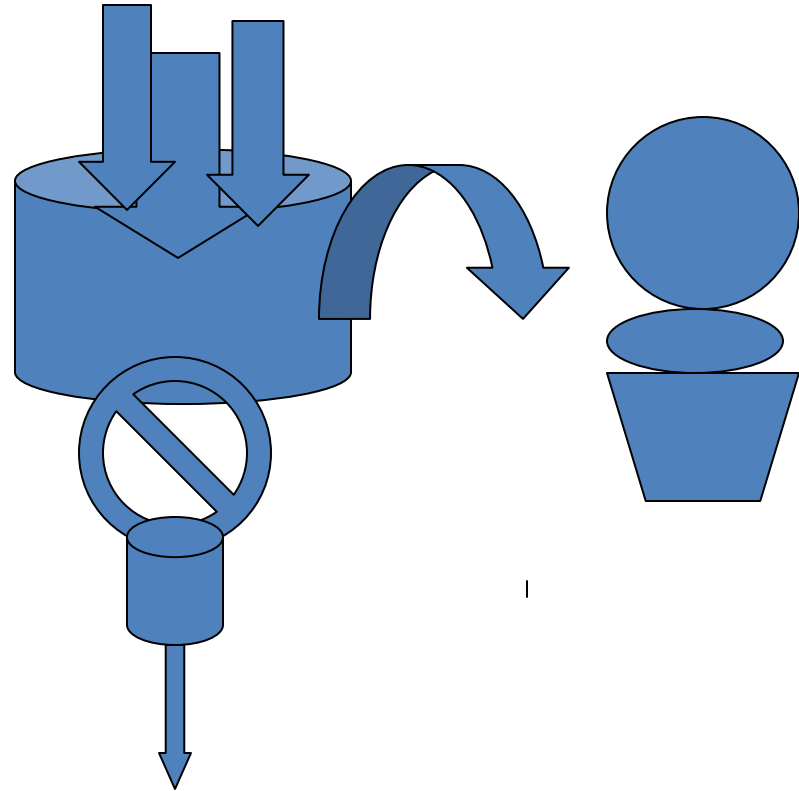
\*\* Higher HDLs--for every 21-mg/dL **increase** in HDL, people are ½ as likely to develop albuminuria  
(*Diabetes Care* January 06)

# What else do the statins do?

- Decrease total cholesterol
- Decrease LDL-cholesterol
- Decrease oxidation of LDL-cholesterol
- Shrink plaques including plaques in the renal artery
- Prevent the formation of new plaques
- Decrease mesangial proliferation
- Decrease vascular inflammation
  
- What's not to love?

# The Diabetic Kidney...hyperglycemia/HTN (*the deadly duo*)

- Hyperglycemia and/or hypertension boost prostaglandins and vasodilate the afferent arteriole
- Hyperglycemia and hypertension increase angiotensin 2 and vasoconstrict the efferent arteriole



Microalbuminuria  
(between 30 mg—300  
mg of alb/g creatinine—  
10-fold > risk of RD &  
CKD)

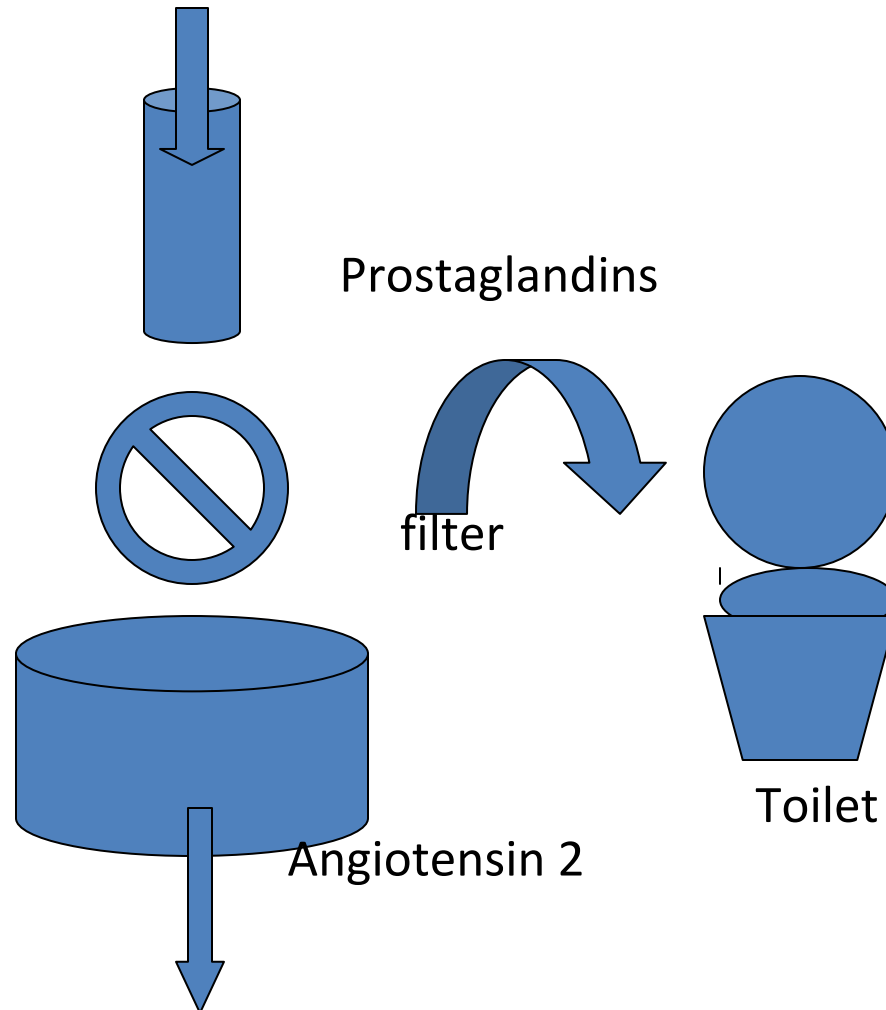
## **“prils” and “sartans” to the rescue (Ace inhibitors and angiotensin receptor blockers or ARBS)—protect kidneys**

- Captopril (Capoten)
- Enalapril (Vasotec)
- Lisinopril (Prinivil, Zestril)
- Perindopril (Aceon)
- Moxepril (Univasc)
- Benazepril (Lotensin)
- Quinapril (Accupril)
- Trandolapril (Mavik)
- Ramipril (Altace)
- losartan (Cozaar), valsartan (Diovan), candesartan (Atacand), telmisartan (Micardis)  
irbesartan—Avapro  
olmesartan—(Benicar)

# NSAIDs (block PGs) + ACE inhibitors (block AT2)

- NSAIDs vasoconstrict the afferent arteriole
- ACE inhibitors block angiotensin 2 vasodilating the efferent arteriole

**Nothin' in,  
everything  
OUT!**



# What else can go wrong with the **blood** supply of the kidney?

- Clamping the aorta above the renal artery (AAA)
- Sudden cessation with a renal artery embolus
- Decreased blood pressure --acute blood loss, hypovolemic shock, heart failure, dehydration, septic shock
- Microthrombosis of glomeruli—DIC (disseminated intravascular coagulation)
- Decreased filtration due to myoglobin from rhabdomyolysis
- Immune complex deposition in the glomerulus triggering the inflammatory response (lupus nephritis)

# Systemic lupus erythematosus

- Immune “complex” disease—Ag/Ab complexes
- Complexes deposit in tissues that are innocent bystanders, including the glomerulus
- 1/250 African American women aged 18-65; 1/1000 Caucasian women

# The anatomy of a nephron—greater detail

- The basic functioning unit of the kidney
- The nephron—1.5 million per kidney in normal **birth weight** individuals

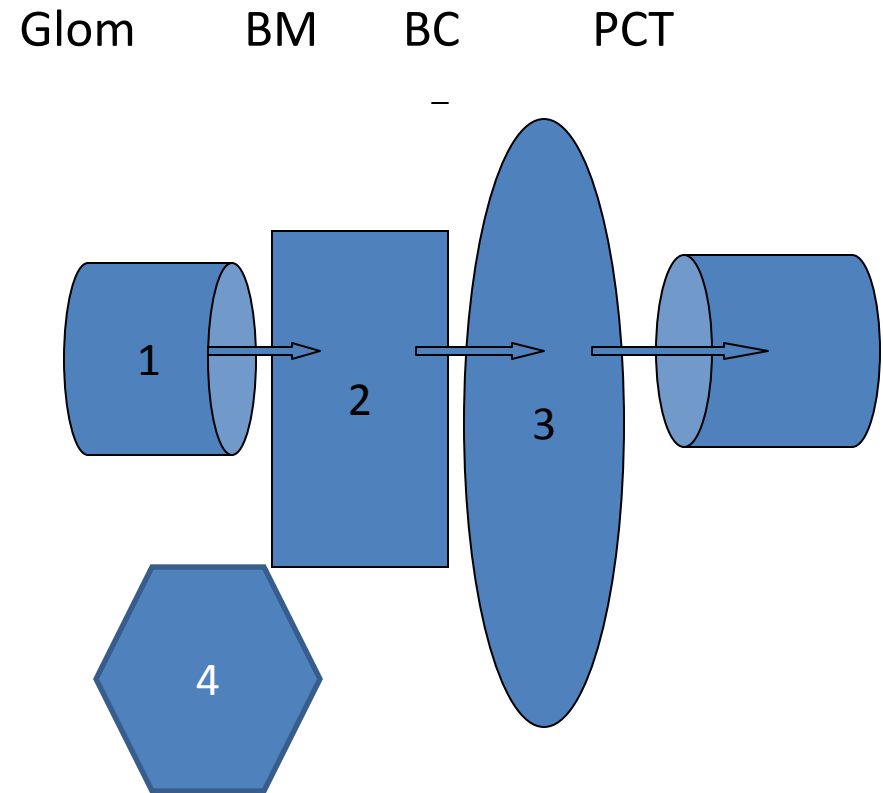
# Premature babies/LBW babies

- Is your risk of hypertension related to the number of nephrons you were born with?
- LBW babies are much more likely to develop hypertension later on in life and it may be due to the fact that they have less nephrons to start with
- Autopsies on patients between 35-59
- 10 kidneys w/ known hypertension; 10 w/ normal BP
- Average number of nephrons in people w/ HBP was fewer than  $\frac{1}{2}$  that of people w/ normal BP
- Couldn't find damaged nephrons or nephrons that had dropped out—suggesting inherited # of nephrons
- (*N Engl J Med* 9 Jan 2003)

# The glomerular filtration membrane

3 layers:

1. The glomerular capillary wall (endothelial cells)
2. The basement membrane (a glycoprotein layer)
3. The fenestrated wall (epithelial) cells of Bowman's capsule into the first part of the tubule (the proximal tubule)
4. Mesangial cells—



# What can go wrong?

- 1) Lupus nephritis
- 2) diabetic nephropathy adults w/ nephrotic syndrome—MM, HBV, SLE causing thickened BM (membranous nephropathy)
- 3) nephrotic syndrome (minimal change nephropathy—90% kids associated w/ IgE-mediated disease)

# A note on the tubules of the kidney...

- The tubules (like Bowman's capsule and the PCT—proximal convoluted tubule) are lined with ***epithelial*** cells
- The epithelial cells are extremely vulnerable to hypoxia
- Without oxygen, the epithelial cells become necrotic and slough into the tubule; clogging the works
- Acute tubular necrosis (ATN)
- Is it possible to recover from ATN? Why?

# Ethylene glycol nephrosis

- Dogs and cats love the sweet taste of antifreeze
- Crystals precipitate in the tubular lumen resulting in intrarenal obstruction, degeneration and necrosis of the lining of the tubular epithelium
- This nephrosis is NOT reversible in dogs and cats

# MAJOR FUNCTIONS OF THE KIDNEY

- Regulation of water, solutes, electrolytes, and acid-base balance
  - 1) urea, creatinine
  - 2) sodium, potassium, calcium, phosphorus
  - 3) hydrogen and bicarbonate

- **If the kidney FAILS...**

Retention of water—edema, weight gain, HTN

Retention of urea, creatinine)

Retention of sodium, potassium resulting in hypertension, hyperkalemia,

Retention of H<sup>+</sup> ions—metabolic acidosis

# Kidney produces the active form of vitamin D, calcitriol

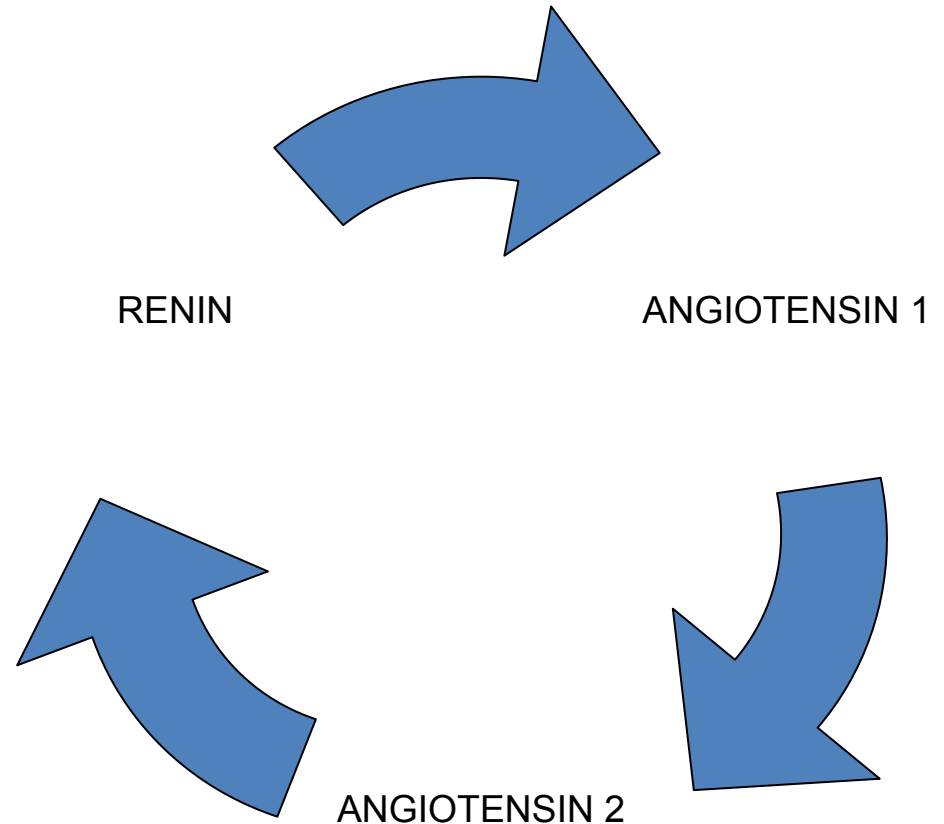
- Vitamin D is necessary for the absorption of calcium from the GI tract
- With increased phosphate retention or decreased calcium absorption the parathyroids increase their production of PTH
- PTH breaks down bone to replace the calcium—secondary hyperparathyroidism
- Renal osteodystrophy

# Major vitamin D deficiency in U.S.

- 10-15 minutes of exposure to sunlight on face, hands, and arms 2-3 days per week is required to synthesize sufficient amounts of vitamin D
- Darker-skinned individuals need more time
- **NO SUNSCREEN**

# The kidney plays an essential role in maintaining blood pressure

- Low volume, low pressure?
- JGA cells in afferent arteriole release renin-- angiotensin 1 (liver) is converted to angiotensin 2 (tissues) via the enzyme ACE;
- Angiotensin 2 runs over to the adrenal cortex to stimulate the release of aldosterone



# What do “Angie” and “Al” do?

- **“Tenses” your “angios”—vasoconstricts**
- **Triggers release of “AL”—aldosterone (from the adrenal cortex to save Na<sup>+</sup> & H<sub>2</sub>O)**

## What else does angiotensin-2 do?

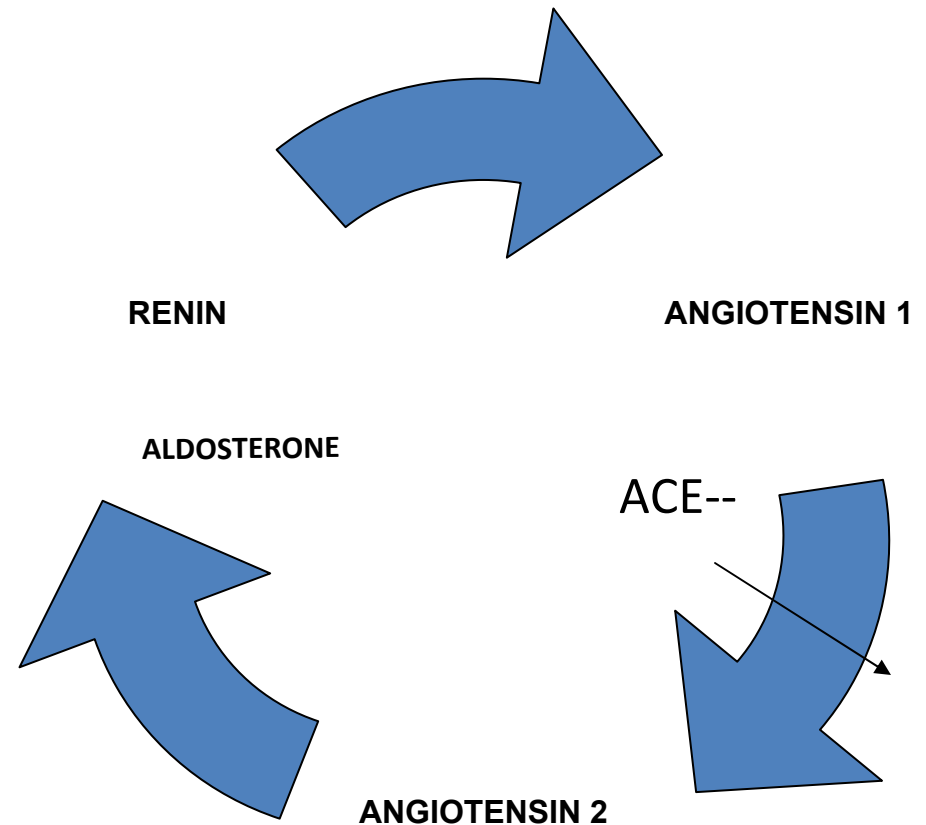
- **Increases inflammation in the arteries**
- **Prothrombotic**
- **Increases tissue resistance to insulin**
- **Potent growth factor—“angie” remodels tissues; Is remodeling a good word? NOT...**

# But NOT...

- In your heart after a myocardial infarction, OR
- In your heart with chronic heart failure, OR
- In your blood vessels with hypertension,
- OR
- In your kidneys with diabetes or hypertension
- “Angie is a bad girl...”

# Enter the “PRILS”—The ACE inhibitors

- Who is “ACE” and why do we want to inhibit him?
- Angiotensin Converting Enzyme (ACE) inhibits the conversion of AT1 to AT2
- ACE inhibitors block the conversion of angiotensin 1 to 2



# The kidney produces erythropoietin

- Essential for the production of RBCs—failing kidney does not secrete erythropoietin
- One of the earliest signs of declining renal function is the presence of anemia
- Anemia has been independently associated with an increased risk of LV dilation, **LVH**, CAD, HF
- The link between heart failure, CKD, and renal failure is known as *cardiorenal anemia syndrome*
- (Ghanasekaran I, Dimitrov H. Primary care management of anemia in chronic kidney disease. *Patient Care* May 2006)

# The end.

- The average person will pass about 11,000 gallons of urine in a lifetime.
- Thanks.