Renal & IV Therapy

Functions of the Kidney:
- Eliminate toxins and waste
- Maintain BP
  - In response to Aldosterone, ADH
  - Think of the whole renin, Ang II pathway
- Stimulate RBC production (remember that the kidneys sense a drop in H&H and this stims the release of erythropoietin)
  - Know that pts will have bone pain if given erythropoietin
- Activate Vitamin D
- Regulate acid-base and electrolyte balance

Failing kidneys can lead to high BP, because they are not able to do their job in regards to the whole maintain BP thing. Also, if your pt’s kidneys don’t work well, you will need to adjust their dosages and meds, and you’ll have to watch fluid intake too! Busy busy!

Diagnostics: KUB and CT
A KUB is an x-ray of the kidneys, ureter, bladder. Your pt will get this when they are on a feeding tube (it must also show the stomach, intestines?). A CT-scan shows cross-sections of the anatomy and involves the pt having to drink a dye that could ultimately cause kidney failure.

Some kidney facts:
- Most of the “stuff” that is absorbed in the kidneys is absorbed at the PCT.
- When the bladder is full of urine it backs up to the loop of henle.
- We have about 2 million nephrons…each one is tiny but there sure are a lot of them!
- AAA (abdominal aortic aneurysm can block off the blood supply to the renal artery…that’s bad news.

The afferent arterioles go IN to the kidney, the efferent arterioles go OUT.

The peritubular capillaries provide nourishment and oxygen to the kidneys. Problems with blood supply (either due to low BP or obstruction at the glomerulus) leads to oxygenation issues of the tubule and you get renal failure. Again…bad news.

The glomerulus is a high-pressure capillary system that pushes fluid into the filtrate. The higher the pressure exerted at the efferent arteriole, the higher the pressure in the glomerulus…this leads to MORE filtrate!

If the filtrate has protein and blood cells in it, this is more bad news for your pt and indicates a problem with the glomerulus.

Each glomerulus filters 0.1 ml which equates to 180 L per day with all of them working together!

The capillaries here are 25x more permeable than systemic capillaries, and they have a huge surface area!

The PCT:
- 60-80% of filtrate is reabsorbed at the proximal convoluted tubule (PCT).
- Of the 65,000 mEq of Na filtered each day, the body only wastes 140 to excretion…wow!
- An osmotic diuretic (such as glucose or mannitol) prevents reabsorption of Na.
Glucose is reabsorbed on a transport system which is fully loaded at blood sugar levels of about 250…anything about that is spilled into the urine. In the old days, they used to monitor glucose with urine testing, but that definitely is not very accurate!

H+ is secreted in exchange for bicarb.

The Loop of Henle:
- The ascending limb pumps Cl, Na out into the interstitium, but it is impermeable to water! This, the interstitial space is becoming more and more concentrated and the filtrate is becoming more dilute.
- 25% of Na is absorbed here.
- The diuretics that work on the loop are POTENT!

The Distal Tubule:
- 3-5% of Na is reabsorbed here.
- Na is exchanged for K under the influence of aldosterone.
- K sparing diuretics work in the distal tubule.

The Collecting Duct:
- If the pores are open (ADH opens those pores!), then water moves from the dilute tubule fluid into the concentrated interstitial space.
- Urine becomes smaller in volume and gets as concentrated as the interstitial space was.

Comparison of diuretics:
With no Diuretic: The tubules do their thing as outlined above
With Furosemide: Furosemide blocks loop reabsorption of Na, and more Na gets in to the distal tubule for exchange with K…as a result the pt loses K.
With Spironolactone: Spironolactone inhibits aldosterone, so there is no exchange of K for Na in the tubule fluid. It is not as potent of a diuretic, but it does spare K.

If a pt’s urine is dilute, then they probably took their diuretic. If it’s amber colored and they tell you they took their Lasix…they are trying to pull a fast one!

How much urine do you need each hour? Well, the short answer is that it’s 20 ml/hr, but we always go with 30ml/hr as the standard for the hospital b/c if people are sick they are likely producing more toxins and these need to be flushed out! There’s a fancy equation that goes like this:

\[
\frac{700 \text{mosm/day} \times \text{day/24 hr} \times \text{liter/1400 mosm} \times \text{1000 ml/liter}}{}
\]

Anatomy review:
- Peristals moves urine forward along the ureters (don’t want it backing up into kidneys!)
- The ureters are innervated, so if someone has kidney stone it HURTS!
- Valves in the bladder do their darndest to keep the urine from backing up into kidneys.
- The bladder holds 1000-1800 mls
- Voiding occurs at around 200-400 mls, and hopefully it completely empties. If not, this is called urinary retention and it leads to UTIs.
- Flushing removes any bacteria, and macrophages search out any that are remaining!

Let’s prevent a UTI! Catheterization always carries with it a risk of a UTI, so we need to do as much as we can to keep this from happening.
- Some catheters are impregnated with antibacterial substances
- Remove the cath as soon as you can…if there is EVER another option, you will always want to go with that option as opposed to leaving in a cath or inserting one in the first place.
- In & Out caths have less risk of infection, so do those if you can.
- Bladder scanning after the pt voids shows you if they have any retained urine…it’s better than putting in a catheter!
- A UTI increases the cost of care by $600 and it’s no fun for the pt either!
Things that cause a UTI besides catherization are:
- Obstruction...for example, pts with BPH essentially have an obstruction caused by the prostate
- Hypotonic bladder (doesn’t contract)
- Female (short urethra, close to anus)
- Older
- Immune suppressed
- Uncircumcised (keep it clean, just be sure to put foreskin back where it belongs!)
- Poor hygiene/lots of sex (a bad combo)

FYI: When your pt's urine is positive for leukocyte esterase then they have a UTI.
Cystitis can be asymptomatic...it is an inflammation of the bladder
If the infection goes to the kidneys, then you'll have systemic effects (fever, increased WBC)

Everything you wanted to know about the MICTURITION REFLEX!
As the bladder fills with urine it will stretch and send a message to the brain. The brain, however, is more patient than the bladder and says “wait!”. So, the bladder waits and fills some more...it sends another message to the brain which still says, “wait!”. So the bladder waits some more. Finally, the brain decides to listen to the bladder (the brain must be a man...they never listen!) and the brain activates the PNS which causes the bladder to contract, and blocks the SNS which relaxes the sphincter...as a result, you pee!

But what if this whole system isn’t working the way it should? Glad you asked!
- Neurogenic Bladder can be either hyperreflexive or hyporeflexive.
- Hyperreflexive bladder often occurs with spinal cord injury and it involves the pt having NO CONTROL of their bladder contraction. The contraction is not coordinated with the relaxation of the sphincter and the bladder does not completely empty. There will be a small bladder volume and reflux of urine back up to the kidney which is bad news...not sure if it’s pyelonephritis or hydornephrosis...both were mentioned.
- Hyporeflexive bladder happens when there is damage to the nerve and the messages do not go where they are supposed to go. The bladder fills and does not empty (ouch!). There will be a large bladder volume and reflux back up to the kidneys, plus a large amount of residual. This condition is similar to what you see in diabetes.

Incontinence
There are several types of incontinence:
- Stress: weakened muscles and support structure d/t multiple pregnancies, jogging and obesity
- Urge: this is caused by infection and irritable bladder
- Overflow: This is the result of hypotonic bladder
- Functional: This is the result of not being able to get to the bathroom. Maybe the person is in restraints, or has a physical limitation

The treatments for incontinence are:
- Strengthen the muscles and tighten the sphincters by doing Kegals (50 a day!), electric stimulation, implants and surgery.
- Timed voiding...I think this trains the bladder and ensures someone doesn’t get so full that they have to rush to the bathroom and possibly not make it
- Medications
  - Oxybutynin (Ditropan): decreases contractility
  - Bethanechol (Urecholine): increases tone
  - Antibiotix
  - B+O (Belladonna and Opium): antispasmotic
Phenazopyridine (Pyridium): analgesic…it turns your pee bright orange

Pads and catheterizations are also another treatment. The goal is to avoid a catheterization as much as possible! Incontinence in and of itself is not a reason to put in a Foley.

Pre-Renal Failure

Pathophysiology:
In pre-renal failure, the kidney system works normally but is excreting a small volume of concentrated urine. Low blood flow leads to decreased GFT, leading to decreased urinary output, which increases concentration, decreases the amount of urine sodium, and an increased BUN/creatinine. The most common etiology of this is a low BP/cardiac output.

Some more info I found online:

- Prerenal ARF is characterized by inadequate blood circulation (perfusion) to the kidneys, which leaves them unable to clean the blood properly. Many patients with prerenal ARF are critically ill and experience shock (very low blood pressure). There often is poor perfusion within many organs, which may lead to multiple organ failure.
- Prerenal ARF Causes: Some of the most notable causes of prerenal ARF are dehydration, heart failure, sepsis (severe infection), and severe blood loss.
- Prerenal ARF is associated with a number of preexisting medical conditions, such as atherosclerosis ("hardening" of the arteries with fatty deposits), which reduces blood flow. Dehydration caused by drastically reduced fluid intake or excessive use of diuretics (water pills) is a major cause of prerenal ARF. Many people with severe heart conditions are kept slightly dehydrated by the diuretics they take to prevent fluid buildup in their lungs, and they often have reduced blood flow (underperfusion) to the kidneys.
- Prerenal ARF Risk Factors
  - Atherosclerosis
  - Blood loss
  - Chronic liver disease
  - Heart disease

S&S:

- Dizziness
- Dry mouth
- Low blood pressure (hypotension)
- Rapid heart rate
- Slack skin
- Thirst
- Weight loss
- Urine output is usually low in people with prerenal ARF.
- The patient also may have symptoms of heart or liver disease.

Prerenal ARF Complications:

Many prerenal ARF patients are critically ill and require admission to an intensive care unit. They may suffer from severe infection, such as viral hepatitis. Decreased perfusion can cause acute organ failure, such as cardiac or liver failure.

Symptoms of heart failure include:

- Dyspnea (shortness of breath)
- Edema (fluid retention and swelling)
- Venous engorgement
Symptoms of liver failure include:
- Confusion, disorientation, stupor
- Sweet, ammoniacal odor

**Prerenal ARF Diagnosis:**
A complete physical examination and a medical history help the physician to diagnose prerenal ARF. In addition, laboratory studies often reveal a high BUN to Cr ratio (BUN:Cr > 20:1), along with abnormal urine chemistry. The physician often must rule out postrenal and intrinsic renal causes of acute renal failure.

**Prerenal ARF Treatment:**
The goal of treatment is to improve of kidney perfusion (blood circulation). This usually involves treating the underlying condition (e.g., infection, heart failure, liver failure). Intravenous (IV) fluids are administered to most patients to treat dehydration.

**Prerenal ARF Prognosis:**
In general, patients with prerenal ARF improve dramatically with intravenous fluids. Urine output increases and renal function improves.

**Glomerular Dysfunction**
The capillary membrane of the glomerulus acts as a filter. When this membrane is not functioning properly it leads to proteinuria, hematuria, hypoalbuminuria and lipidemia. Decreased blood flow to the glomerulus leads to volume overload and tubular dysfunction.

**Glomerulonephritis**
can occur d/t Ab-Ag complexes after a pt has strep throat, an autoimmune disease such as goodpasture’s or lupus, and diabetes. It i s a type of kidney disease that damages your kidneys’ ability to remove waste and excess fluids. Also called glomerular disease, glomerulonephritis can be acute or chronic. If glomerulonephritis occurs on its own, it's known as primary glomerulonephritis. If another disease, such as lupus or diabetes, is the cause, it's called secondary glomerulonephritis. Treatment depends on the type of glomerulonephritis you have.

**S&S of glomerulonephritis**
Signs and symptoms of glomerulonephritis may depend on whether you have the acute or chronic form, and the cause. Your first indication that something is wrong may come from symptoms or from the results of a routine urinalysis. Signs and symptoms may include:
- Cola-colored or diluted, iced-tea-colored urine from red blood cells in your urine (hematuria)
- Foamy urine due to excess protein (proteinuria)
- High blood pressure (hypertension)
- Fluid retention (edema) with swelling evident in your face, hands, feet and abdomen
- Fatigue from anemia or kidney failure
- Less frequent urination than usual

**Causes:**
Each of your kidneys contains approximately 1 million tiny filters (glomeruli), which attach to the opening of a small fluid-collecting tube (tubule). Each glomerulus and tubule form a nephron, the functional unit of the kidneys. The glomeruli filter your blood as it passes through your kidneys, and the filtered blood returns to your bloodstream. The tubules modify what the glomeruli filter by saving needed substances, such as protein. The waste goes to your bladder as urine through a tube (ureter) from each kidney and passes out of your body when you urinate. Glomerulonephritis — an inflammation of the glomeruli — can damage your kidneys so that they lose their filtering ability, allowing dangerous levels of fluid and waste to accumulate in your body (called kidney failure) and depriving your bloodstream of protein, which is excreted in your urine. Often the cause of glomerulonephritis is unknown. Known causes include:
Infections
- Post-streptococcal glomerulonephritis. Glomerulonephritis may develop after a strep infection in your throat or, rarely, on your skin (impetigo). Post-infectious glomerulonephritis is becoming less common in the United States, most likely because of rapid and complete antibiotic treatment of most streptococcal infections.
- Bacterial endocarditis. Bacteria can occasionally spread through your bloodstream and lodge in your heart, causing an infection of one or more of your heart valves. Those at greatest risk are people with a heart defect, such as a damaged or artificial heart valve.
- Viral infections. Among the viral infections that may trigger glomerulonephritis are the human immunodeficiency virus (HIV), which causes AIDS, and the hepatitis B and hepatitis C viruses, which primarily affect the liver.

Immune diseases
- Lupus. A chronic inflammatory disease, lupus can affect many parts of your body, including your skin, joints, kidneys, blood cells, heart and lungs.
- Goodpasture's syndrome. A rare immune lung disorder that may mimic pneumonia, Goodpasture's syndrome causes bleeding (hemorrhage) into your lungs as well as glomerulonephritis.
- IgA nephropathy. Characterized by recurrent episodes of blood in the urine, this primary glomerular disease results from deposits of immunoglobulin A (IgA) in the glomeruli. IgA nephropathy can progress for years with no noticeable symptoms. The disorder seems to be more common in men than in women.

Vasculitis
- Polyarteritis. This form of vasculitis affects small and medium blood vessels in many parts of your body, such as your heart, kidneys and intestines.
- Wegener's granulomatosis. This form of vasculitis affects small and medium blood vessels in your lungs, upper airways and kidneys.

Conditions that cause scarring of the glomeruli
- High blood pressure. Damage to your kidneys and their ability to perform their normal functions can occur as a result of high blood pressure. Glomerulonephritis can also cause high blood pressure because it reduces kidney function.
- Diabetic kidney disease. Diabetic kidney disease (diabetic nephropathy) can affect anyone with diabetes. Diabetic nephropathy usually takes years to develop. Good control of blood sugar levels and blood pressure may prevent or slow kidney damage.
- Focal segmental glomerulosclerosis. Characterized by scattered scarring of some of the glomeruli, this condition may result from another disease or occur for no known reason.

Chronic glomerulonephritis sometimes develops after a bout of acute glomerulonephritis. In some people there's no history of kidney disease, so the first indication of chronic glomerulonephritis is chronic kidney failure. Infrequently, chronic glomerulonephritis runs in families. One inherited form, Alport syndrome, may also involve hearing or vision impairment.

Complications of glomerulonephritis:
- Acute kidney failure...loss of fxn in the filtering part of the nephron may cause waste products to accumulate too rapidly. The pt will need emergency dialysis.
- Chronic kidney failure...is a VERY serious compication where the kidneys gradually lose function. Kidney function at less than 10% of normal capacity indicates end-stage kidney disease, which usually requires dialysis or kidney transplant.
- High blood pressure...damage to the kidneys and the resulting buildup of wastes in the bloodstream can raise BP.
Treatment varies whether you have the acute or chronic form of the disease. Some cases of acute glomerulonephritis (especially those that follow strep), can improve on their own. Other tx include:

- Controlling high blood pressure
  - Control salt intake
  - Diuretic
  - ACE inhibitor
  - ARB
- Treat the underlying cause
  - Abx to treat strep or bacterial infection
  - Corticosteroids and immune-suppressing drugs for lupus or vasculitis
  - Fish oil supplements for IgA nephropathy (currently being studied)
  - Plasmapheresis sometimes used to treat Goodpasture’s.
- Treat kidney failure
  - Temporary dialysis may be needed for acute forms of the disease.
  - The only long term therapies are dialysis and transplant.

**Nephrotic Syndrome** is a disorder caused by damage to the small blood vessels in your kidneys that filter waste and excess water from the blood. When healthy, these small vessels keep protein from seeping into your urine and out of your body. When damaged, they don’t do this very well and protein leaks out of the blood and can lead to edema. Nephrotic syndrome can increase the pt’s risk for infections and blood clots, so be aware of that!

**S&S of nephrotic syndrome:**
- Edema, particularly around eyes, ankles, feet
- Foam in the toilet water d/t protein in the urine
- Weight gain d/t excess fluid retention
- Loss of appetite
- Vomiting

**Causes of nephrotic syndrome:**
Nephrotic syndrome is caused by damage to the tiny blood vessels (glomeruli) of your kidneys. Your kidneys are two bean-shaped, fist-sized organs situated at the small of your back, just below your rib cage, one on each side of your spine. Blood enters your kidneys through arteries from your aorta, the large artery that carries blood away from your heart. Each kidney contains approximately 1 million glomeruli (the singular word form is glomerulus), each of which is attached to the opening of a small fluid-collecting tube (tubule). Each glomerulus and its tubule form a nephron, the functional unit of the kidneys.

A filtering role

The glomeruli filter your blood as it passes through your kidneys. After being filtered by the glomeruli, blood travels through veins in your kidneys back to your bloodstream. The filtered substances, after being modified by the tubules, go through a tube from each kidney (ureter) to your bladder and pass out of your body when you urinate.

Every day about 2 quarts of water, along with waste products and electrolytes, leave your body as urine. When your kidneys lose their filtering ability, dangerous levels of fluid and waste accumulate in your body, a condition known as kidney failure.

What happens in nephrotic syndrome

Healthy glomeruli keep blood protein (mainly albumin) — which is needed to maintain the right amount of fluid in your body — from seeping into your urine. When damaged, glomeruli often lose this ability. Loss of blood protein leads to nephrotic syndrome, which is characterized by:
- High levels of protein in your urine (proteinuria)
- Low levels of the blood protein albumin (hypoalbuminemia)
- Elevated blood levels of cholesterol and triglycerides
- Swelling (edema)

Many possible causes

Many disorders can cause glomerular damage and lead to nephrotic syndrome. The following medical conditions account for most cases of nephrotic syndrome:

- Minimal change disease. The most common cause of nephrotic syndrome in children, this disorder results in abnormal kidney function, but when the kidney tissue is examined under a light microscope, it appears normal or nearly normal. The cause of the abnormal function typically can't be determined.
- Focal segmental glomerulosclerosis. Characterized by scattered scarring of some of the glomeruli, this condition may result from another disease, a genetic defect or occur for no known reason.
- Membranous nephropathy. This kidney disorder is the result of thickening membranes within the glomeruli. The exact cause of the thickening isn't known, but it's sometimes associated with other medical conditions, such as hepatitis B, malaria, lupus and cancer.
- Diabetic kidney disease. Diabetes can lead to kidney damage (diabetic nephropathy) that affects the glomeruli, particularly in people with diabetes that's poorly controlled or people who have high blood pressure.
- Systemic lupus erythematosus. This chronic inflammatory disease can lead to serious kidney damage.
- Amyloidosis. This disorder occurs when substances called amyloid proteins accumulate in your organs. Amyloid buildup often affects the kidneys, damaging their filtering system.

Complications

- Blood clots. The inability of the glomeruli to filter blood properly can lead to loss of blood proteins that help prevent clotting. This increases your risk of developing a blood clot (thrombus) in your veins.
- High blood cholesterol and elevated blood triglycerides. When your blood level of the protein albumin falls, your liver makes more albumin. At the same time, your liver releases more cholesterol and triglycerides.
- Poor nutrition. Loss of too much blood protein can result in malnutrition. This can lead to weight loss, but it may be masked by edema.
- High blood pressure. Damage to your glomeruli and the resulting buildup of wastes in your bloodstream (uremia) can raise your blood pressure.
- Acute kidney failure. If your kidneys lose their ability to filter blood due to damage to the glomeruli, waste products may build up quickly in your blood. If this happens, you may need emergency dialysis — an artificial means of removing extra fluids and waste from your blood — typically with an artificial kidney machine (dialyzer).
- Chronic kidney failure. Nephrotic syndrome may cause your kidneys to gradually lose their function over time. Kidney function at less than 10 percent to 15 percent of normal capacity is considered end-stage kidney disease, which usually requires dialysis or a kidney transplant.
- Infection. Although it's not clear why, people who have nephrotic syndrome are at an increased risk of infection, such as pneumonia.

Tests and Dx

Urinalysis  
Blood test  
Kidney biopsy

Tubular Dysfunction

This problem is caused by damage to the tubular cells and the osmolarity of the urine then equals the osmolarity of the plasma. The result is oliguria, because there is no concentration gradient to pull fluid into the tubule. The pt will present with increased BUN/creatinine. Clinical examples of tubular dysfunction are
nephrotoxins such as gentamicin, contrast dye (be careful of those CT scans!), ischemia which has something to do with the progression of preglomerular or just regular ol’ glomerular disease, pyelonephritis (back up of urine into the kidneys...this hits the long loops of Henle first).

**Pyelonephritis** is a common disease of the kidney. It is associated with cystitis and reflux. The S&S are flank pain, fever, WBC/bacteria in urine. The predisposing factors are neurogenic bladder (hypotonic and hypertonic) and obstructive uropathies such as BPH, a kinked catheter, foreign objects, tumors and stones.

**Calculi**
The most common calculi (kidney stones) are calcium oxalate or calcium phosphate stones and magnesium stones. Magnesium stones are also called “struvite”. The stones are very painful to pass, but most can dissolve or pass with increased fluid intake and by altering the pH and electrolytes of body fluids/urine. You can also limit oxalate, Na, Ca and purines. If you have uric acid stones, then you’d alkalanize urine to get rid of them.