Renal Structure
The kidneys are a pair of bean shaped organs that lie outside the peritoneal cavity between the T-12 and L-3. The right is slightly lower than the left because the liver is in the way (the liver is HUGE). The kidney is made up of three layers, the cortex, medulla and pelvis. The cortex is the outer layer, the medulla is where the nephrons are, and the pelvis is the very inner part where urine collects. Note that the renal artery is second aorta off the heart (first is hepatic)...they get a large blood supply and this is why urine output is an indicator of organ/tissue perfusion.

Renal Perfusion...a huge deal! Love to be flooded with fluid all the time!
- Kidneys are perfused with 1000 to 1300 ml of blood per minute
- This amounts to 20 – 25% of cardiac output (another 25% goes to hepatic). These two organs get a LOT of blood!
- Recall that GFT = glomerular filtration rate...it is the rate at which blood flow perfuses through the renal artery into the filtration system of the kidney.
- A sufficient GFR is needed for removal of waste products (GFR= how much blood going into the kidneys and how much filtration is going on) > or < 60 ml/min...what we watn to know is that GFR less than 60, then we have to start adjusting meds and being careful about contrast dye...that means there is renal insufficiency.
- Feedback mechanisms are designed to keep GFR up despite changes in circulatory status. The body will sacrifice a lot of things, but will not sacrifice the kidneys (the body will even sacrifice the brain in favor of the kidneys!)
    - SNS maintains GFR. Recall that SNS is responsible for heart rate and vasodilation/vasodilation. Vasoconstriction and tachycardia will keep kidneys perfused...these are SNS responses! If the body has a lowered perfusion state, the kidneys will try to compensate (as in shock).

Micro Structure
- The working unit of the kidney is the nephron whose primary function is to filter blood. There are about 1 million in each kidney. Each nephron consists of three parts:
    - glomerulus for filtration (filters blood as it comes through the Juxtoglomerular Apparatus)
    - tubular components (designed to equilibrate things like K, Na, glucose, etc...)
    - Collecting duct (purpose of collecting filtrate and sending it to renal pelvis and out through ureter)

Kidney Function
- Filtration and removal of wastes (extra nutrients, glucose, etc...)
- Fluid and electrolyte balance
- Acid Base Balance (works to maintain that normal range of pH...a very narrow window! 7.40-7.45!
- RBC production (secrete erythropoietin to stim RBC production)
- Calcium absorption (kidneys do this, too!)

Glomerulus
The glomerulus is a compact tuft of capillaries encased in Bowman's Capsule. It is essentially a series of membranes allowing for filtration of blood that is entering the nephron. It is a high-pressure, semi-permeable capillary bed. So, for adequate filtration to occur, there must be adequate blood volume in the intravascular space and adequate hydrostatic pressure from the cardiac output and vascular resistance.

Alterations in these mechanisms occur in chronic diseases (diabetes, HTN, kidney disease). When damaged, they no longer filter appropriately...so when your pt has long-term disease you need to look at kidney function...they will be spilling sugar and protein (no longer filtering)...this is a sign of strain on the kidneys d/t DM and HTN. Tells us some of the nephrons are damaged!

Glomerular Filtration Rate
- Measures the plasma volume that can be cleared of any given substance
- Used as an indicator of adequacy of renal function
- Altered by diseases that change plasma flow or permeability of membrane
- Normal GFR is 180 L per day. Minimum GFR is 60 mL / hour (will be on the chem panel <60 or > 60). If < 60 we have to adjust meds and avoid contrast material.
- Tells us how well we can clear any given substance (a med, a nutrient, acid, etc...)

Tubules of the Nephron:
The tubules are responsible for adjusting all the electrolytes to maintain homeostasis. (Reabsorption/Secretion) Ex: If filtrate is delivered to G and filters out a bunch of Na, the filtrate goes out to the tubule and now the Na level in the body has dropped a little bit, the tubule will send some of that Na back into circulation (reabsorption).

- Tubular Reabsorption
  - Filtrate returned to the blood supply
- Tubular Secretion
  - Substances secreted into tubules to be excreted
- 4 segments
  - Proximal Convoluted Tubule
  - Loop of Henle (important...the loop where things like Lasix work to Increase urine output "Loop Diuretics")
  - Distal Convoluted Tubule
  - Collecting tubule

Tubular reabsorption is accomplished in the proximal convoluted tubules of the kidneys. Reabsorption occurs as a result of active transport and passive transport. Tubular secretion is the process by which substances like K, H and abx are secreted into the tubules to be excreted in the final stage of urine formation. The final concentration of dilution of urine occurs in the distal tubules and collecting ducts that lead to the bladder. The volume of urine excreted should be about 1500 mL per day. Less tahn 400 mL per day is considered renal failure.

Urine Formation: A Three step process (body constantly makes adjustments throughout)

- Filtration : filters out excess potassium, H+ ions, Calcium, glucose, Phosporous etc.
- Reabsorption: calls back electrolytes that are needed
- Secretion: sent through to the collecting ducts to be emptied into the ureter and bladder

Elimination Function of the Kidney
Every substance has its own clearance rate (glucose, K, meds, etc...). Two conditions that can increase renal blood flow and GFR (so we can get rid of those)

- Elevated protein content in the blood
- Elevated glucose content in the blood (ex: polyuria in DM is the body trying to get rid of sugar..does this via vasoconstricion/HTN...explains why DM can cause HTN!)

Urea Elimination
Urea is the end product of protein metabolism (eat chicken, liver metabolizes it and the byproduct is urea...when you eat a lot of protein the urea is going to be high, when liver not working, urea will be low). A normal adult produces 25 – 30 g/ day of urea. Urea in the blood increases with high protein diet, muscle breakdown, GI bleeding (if bleeding into stomach, stomach digests that and sends it to liver to be metabolized), and dehydration.

The kidneys filter and reabsorb to maintain a normal BUN of 8-20. During dehydration, BUN may go up due to a decreased GFR and decreased clearance of BUN. Note that BUN decreases with malnutrition and liver failure as the body no longer metabolizes proteins efficiently and urea is not formed.

Drug Elimination
Drugs are selectively filtered in the glomerulus...they can be reabsorbed or secreted as well (can be sent out and pulled back in). Drugs that are not bound to plasma proteins are filtered (they leave very quickly...fat soluble ones don't get filtered out so you have to watch toxic levels of ADEK). Other drugs are detox'd in the LIVER.

Fluid / Electrolyte Balances.
In addition to being adjusted during filtration/secretion stage...are also affected by hormones: aldosterone, anti-diuretic hormone and atrial naturetic peptide (ANP). Baroceptors and chemoreceptors detect changes and initiate the secretion of the hormones.

Aldosterone
Aldosterone is secreted by the Adrenal gland (it is a hormone) in response to fluid deficits...it prompts sodium reabsorption (where sodium goes, so goes water...where Na goes, K goes in the opposite direction.) So it hangs on to Na and Water, but gets rid of K (secretes it into collecting tubule). If we don't have enough Aldosterone, then no Na is reabsorbed and we lose a lot of water in the kidneys. We secrete Aldosterone when we need to retain water.

What happens when Na is low? decreased mentation, confusion, hallucinations and then twitching all the way to seizures. Not really a factor of how low Na is, but how FAST it dropped. Some pt can live with a low sodium...if it gradually dropped off then they may be asymptomatic. When it drops FAST, this causes substantial fluid shifts leading to swollen brain. (ex: Wee Wii contest) And what happens when K too low? Scary changes in contractility.

Anti Diuretic Hormone
ADH is produced/triggered by the hypothalamus and secreted by the posterior pituitary. It enhances the ability of water to follow sodium as it is excreted or reabsorbed. The release of ADH is stimulated by baroreceptors and osmoreceptors that sit in the carotids. (Note that if the carotids get lined with plaque, then these receptors have a hard time doing their job.) When a baroreceptor senses low pressure, it stimulates production of ADH so that the body hangs on to fluid. When the osmoreceptor sense the blood is too concentrated (high osmolarity), it inhibits production of ADH. What is going to happen to urine output with ADH release? It goes down!

Atrial Naturetic Peptide
ANP is synthesized in the muscle cells of the atrium (BNP is synthesized in walls of ventricle). It is released when the atria is stretched, →

- Vasodilation of afferent and efferent arterioles
- Increased renal blood flow
- Increased GFR (wants to get rid of fluid when preload is too high...this happens with CHF to help compensate)
- Inhibits Aldosterone and ADH at the same time (aldosterone conserves water, ADH conserves water)

We can measure BNP and this sometimes tells us if pt is in congestive heart failure. (will be elevated with CHF.)

Acid Base Balance (HCO3, Phosphate, Ammonia)

- North American diet results in 40 – 80 mMol of H+ ions each day
- The kidney is the only pathway for eliminating H+ ions
- The ability of the kidneys to excrete H+ ions depends on buffers
  - HCO3 - bicarbonate (H ions binds with bicarb in urine filtrate...forms CO2 and H20)
  - HPO4 - phosphate (phosphate ions are metabolic end products that bind with H and are excreted)
  - NH3 - ammonia (a byproduct of liver fxn...if liver not working well, ammonia will build up)
- Ex: Lactulose is given as a laxative...look at labs and you'll often see that liver enzymes will be elevated, so lactulose is given to get rid of ammonia that is building up....so a double-whammy.
- When H ions bind up with something like bicarb, sometimes the body will send a lot of CO2 back into the system, and the CO2 goes into the lungs and we get rid of it that way. This explains how the body attempts to compensate for acid/base balance.
- Recall that potassium tends to be high when the body is acidotic. This is because when acidosis occurs, the kidneys will increase the excretion of H and decrease the elimination of potassium so there is a resultant increase in serum potassium levels. Secondly, the H will be forced into the cells and the K will come out of the cells in exchange (both are cations so an exchange MUST take place to maintain the electrical properties of the cell).
- So, in summary...H that is formed in the body is sent to the kidneys to be excreted. The kidneys excrete some of it by linking it up with phosphate and ammonia and sending it away. The rest of it binds with HCO3 to form carbonic anhydrase, then reabsorbs it and sends it to the lungs, where carbonic anhydrase (H2CO3) is broken down to H20 and Co2 and then lungs then blows it off. EXCEPT for when the lungs cannot do their job (hypoventilation, decreased perfusion, COPD, pneumonia, etc...)

<u>Endocrine Function of Kidney: Renin – Angiotensin, Erythropoietin, Vitamin D</u>

Renin - Angiotensin (secreted by kidney when GFR is low)
Renin plays an important role in BP regulation. It is stored in the Juxtaglomerular cells (JG cells)

- ***Renin*** is released
  - in response to a decrease in renal blood flow (GFR)
  - Change in composition of DCT fluid
  - Sympathetic nervous stimulation
- The pathway: Renin acts to convert a circulating plasma protein called ***angiotensin to angiotensin I.*** This is circulated and in the lungs encounters angiotensin converting enzyme. Angiotensin I is converted to ***Angiotensin II*** in the lungs (Ang II is a potent vasoconstrictor). It acts directly on kidneys to decrease Sodium and Water excretion. It also stimulates aldosterone to be released from adrenal gland. (more water conservation as well as Na)

Erythropoietin

EPO is a hormone that regulates and stimulates red blood cell production in bone marrow. It is stimulated by:

- Tissue hypoxia (Ex: COPD pt is chronically hypoxic, so erythropoietin is being pumped out all the time, so always making RBCs...so a lot of times you'll see COPD pt with Hgb in 17-18, and Hct in 50 range = polycythemia vera)
- Anemia
- High altitudes
- Impaired oxygenation d/t cardiac or pulmonary disease
- Persons with ESRD are often severely anemic. They do not make erythropoietin (Hbg 7, Hct < 20 range)

Vitamin D

Activation of Vitamin D occurs in the kidneys. It is responsible for calcium absorption from the GI tract, and regulates calcium deposition in the bones. Ex: ESRD patients have very brittle bones.

<u>Tests of Renal Function. How do we know kidneys are working well?</u>

Urinalysis

- Urine is 95% water, and 5% solutes
- Normally produce 1.5 liters /day
- Urine should NOT contain
  - Plasma proteins
  - Blood
  - glucose
- pH can vary widely
- SpG 1.010 – 1.025 (the higher the number, the more concentrated the urine = dehydration) 1.000 is water!
- pt with proteinuria who is DM, is about 5 years from dialysis.
- A consistent SpG of 1.010 indicates renal failure...the kidneys no longer have the ability to concentrate the urine, so the urine is the same SpG as plasma.
- Want to know how many epithelial cells in the urine...if we don't do a "clean catch" effectively, the epithelial cells will end up in the urine, so the leukocyte that shows up is contaminate...so if you see leukocyte is high, but also epithelial is high then throw it out...the sample is no good.
- A UTI is indicated by the presence of leukocytes and WBCs in the urine.
- A gram negative bacteruria is indicated by positive nitrites with leukocytes.
- Casts indicate increased proteinuria
- RBS should be negative or rare

Blood Urea Nitrogen

Urea is a byproduct of protein metabolism that is formed by the liver and eliminated by the kidneys. It is influenced by protein intake, GI bleeding and hydration. It is less specific for renal function b/c it can go down when nutritinal intake is low or when liver fxn is decreased. Creatinine is the more specific test for kidney fxn.

Creatinine (the gold standard for kidney function test)

Creatinine is a product of creatine metabolism in muscle formation...it reflects GFR (which is a very important thing for us to know). Its release is relatively constant in most people.

- Generally the norm is 0.5 to 1.3 mg/dl (as it climbs to 1.5 you are looking at renal insufficiency)
- There is an age related decline in renal function (so CR will go up a bit with age)
- When creatinine doubles – renal function is ½ of normal. No bueno!

- The picture to the right shows the relationship between creatinine and GFR.

GFR
Can be calculated from creatinine, which is a direct measure of GFR. Cr is not reabsorbed ever ever ever...once it's filtered out it's gone and it is 100% cleared!

- Generally GFR needs to be no less than 60 ml / min
- If the measure drops below 60, we need adjust medication dosages, avoid contrast dyes and monitor closely.

BUN/ CR ratio.
This is how we decide if kidneys poorly perfused or kidney failure..so very important.

- Should be 20 :1 NORMALLY
- Bun 10, Cr 0.5 = 20:1
- If the BUN / Cr ratio is high
  - i.e Bun 20, Cr 0.5 = 40 :1
  - Prerenal condition from dehydration (high ratio = dehydration)
- If the BUN/Cr ratio is low
  - i.e BUN 30, Cr 3.0 = 10:1
  - Intrarenal failure (low ratio = kidney is damaged and not working well)

Chemistries with kidney dysfunction

- Sodium : up or down (depends on type of renal failure and stage)
- Potassium: up
- Calcium: down (Vit D system not working)
- Phosphorous: up (b/c ca goes down. If Ca and Ph both down then nutritional))
- Uric Acid: up
- pH: down (b/c can't buffer H ions)

Lets Review

- What is the functional (working) unit of the kidney? Nephron
- Fluid and solutes are moved from the vascular system into the tubular system of the nephron by? Secretion
- How does the kidney increase blood pressure? Renin-Ang pathway leads to vasoconstriction, also Aldosterone
- What does aldosterone do? retains fluid, retains Na, secretes K
- ADH retains: water.
- The renal parenchyma is where? Basically the tissue inside the kidney (a parenchymal hemorrhage is a small amount, a tsp or two...not a whole body hemorrhage)
- What percentage of cardiac output comprises the total renal blood flow? 20-25%
- A receptor that increases the blood pressure by increasing production of ADH is? Baroreceptor
- What is an osmoreceptor? A receptor that senses osmolality / concentration

<u>Renal Pathology</u>
Pylonephritis
Pylonephritis is an infection of the kidney caused by:

- Unresponsive lower UTI
- Urinary retention
- Ureteral Obstruction (calculi, prostate hypertrophy, pregnancy, urinary catheter)

Pylonephritis: Signs and Symptoms

- Flank pain (just behind abdomen, along the side...the soft area below the ribs). Can be really painful, constant.
- Nausea and vomiting...get dehydrated very quickly which doesn't help kidneys
- Fever, 102-103...feel awful!
- Malaise
- Fever
- Chills
- Elevated WBC

Pylonephritis: Nursing Diagnoses

- Pain
- Altered body temp – fever
- Fear
- Altered fluid / Electrolytes: dehydration
- Infection (could become urosepsis which is bad)

Pylonephritis: Nursing interventions (usually clears up within 48 hours...no long-term damage unless keep getting it)

- Vital signs including temp
- Intake and output
- Specimen collection: clean catch urine / cath specimen
- Rehydration with IV fluids
- Monitor electrolytes and renal function (usually, pylonephritis does not affect fxn)
- Monitor WBC and differential
- Pain medications
- Antiemetics so we can keep fluids in them
- Tylenol for fever
- Abx (cipro, levaquin, tequin)

Glomerulonephritis (often caused by infection that gets out of hand)

Glomerulonephritis is Inflammation of glomerular capillaris that impairs kidneys ability to filter urine. It accounts for 10-15% of glomerular disease, and it is caused by:

- Infections – bacterial endocarditis, viral infs
- Immune diseases – SLE
- Vasculitis
- HIV
- Hep B and C

Glomerulonephritis: Signs and symptoms. Pt will be very ill in acute phase...

- Hypertension (capillaries in glomerulus are swollen and inflammed so kidneys not getting eprfused, leads to HTN)
- Cola colored urine (almost foamy also)
- Periorbital edema (also hands and feet)
- Note: A lot of times, pt can be a college student who is VERY busy...gets sick and doesn't take care of illness....two weeks later shows up with glomerulonephritis. So, see the doc if you have a viral or bacterial infection! Have to be very careful about how this person is going to take care of themselves for the next few months...fluid and sodium restriction, plus diuretics to take off fluid. Can take 6-8 weeks to get them turned around...during that time, very low activity level.

Urinary Calculi (kidney stones)

- Kidney stones (or any where else in the urinary tract)
- Males > females (3:1)
- Incidence higher in warmer regions (maybe dehydration is related)
- 20 – 55 years of age is usual
- Whites > afro americans
- 10% recurrence

Urinary Calculi: Pathophysiology

- Calcium makes up most stones. They typically form in the kidney when there is an infection that is caused by a bacteria that secretes urease.
- Struvite is made up of magnesium and phosphate and ammonium (usually a staghorn-type stone...very painful, has pointy things on it...they do not pass and you have to go in after them.)
- Stone forms when concentrations of urinary constituents exceed solubility

- Struvite stones form in upper UTI ...take longer to move out...can't pass on their own usually, will probably need surgery or the sound-wave therapy to break it up.
- Certain meds can occasionally cause stone formation (hydrochlorothyazide, acyclovir, indinavir perhaps)
- Kidney stone can sit up in kidney for months or years and not cause problems...once it moves to ureter it becomes instantly and very very painful...
- Stone blocks urine and kidney becomes "boggy"...hydronephrosis.
- Also can cause infection in the kidney...so the pt will be on abx.

Urinary Calculi: Signs and Symptoms

- Sudden onset – unilateral flank pain
- Waxing and waning = renal colic
- Nausea and vomiting d/t pain (vagal response)
- Pain radiates to abdomen and ipsilateral testicle in men
- CBCs and chems will usually be normal
- UA will show hematuria (but no 100% of time)
- Gold standard for detection = CT urogram
- KUB will show kidney stone, but not ureteral or bladder stones very well at all. A renal ultrasound can also be done, but it is not as good. Go with the CT.
- May develop signs and symptoms of infection (maybe a low-grade temp, but will always get prophylactic abx)
- Pain instantly goes away when stone passes.
- ex: Pt admitted to unit at 1:30 am with stone...in the ER they've gotten meds for pain. They are still uncomfortable and get dilaudid...they pass stone 45 minutes later...now they've got all this dilaudid on board..and no stone. What happens to them? They stop breathing! The pain level elicits the SNS response that keeps you breathing and awake, but once that stone passes and the pain is gone, the drug is still on board..the pt is at risk for respiratory depression, so you need to keep an eye on this pt.

Urinary Calculi: Nursing Diagnoses

- Acute pain
- Anxiety
- Fluid and electrolyte imbalances
- Infection
- Dehydration

Urinary Calculi: Nursing Care

- Pain control (opiates and anti-inflammatories). Toradol has been used most recently...some evidence that toradol dilates the ureters allowing the stone to pass. It is an anti-inflammatory that problably decreases swelling in the ureter.
- Anti-emetics
- IV fluids (not trying to rehydrate them...this would make kidney more hydornephrotic)
- Patient teaching regarding prevention (drink more water, lemonade and fruit juice, eliminate grapefruit juice and soda, decreasing animal proteins, watch out for nuts, chocolate, tea and strawberries, spinach and rhubarb)
- Prep patient for procedural intervention
    - ESWL (sound waves are aimed at the stone to break it up)
    - Cystoscopic stone removal

<u>Renal Trauma: Laceration: Contusion: Hematoma</u>

Renal Trauma

- Kidneys are most common organ in urinary tract to be injured, they are not very well protected.
- Usually blunt force (football players getting hit from behind, seatbelt injury in MVA)
- Can be GSW or knife...hematuria will develop if the injury reaches the calyces or renal pelvis.
- May be difficult to detect, especially with blunt trauma
- Occurs in 15 – 40% of abdominal trauma

Renal Trauma: Pathophysiology
- Parenchymal laceration (cut through the kidney itself...which is not good!)
- Disruption of renal artery (recall that this artery takes 20-25% of CO...you're going to lose a LOT of blood very quickly into peritoneal space)
- Disruption of collecting system (pulled away from ureter leading to urine in peritoneal space)
- Comminuted renal fracture (broken apart...ouch!...usually requires removal of the kidney)
- All require surgery

Renal Trauma: Signs and symptoms
- Hematuria – gross or microscopic (don't worry too much about microscopic)
- Lumbar and/or abdominal pain
- Rigidity of anterior abdominal wall (from peritoneal irritation d/t blood in the space)
- Local tenderness (percuss over area)
- Accompanying injuries that will increase your suspicion for kidney injury: rib fx, pelvic fx, vertebral injury

Renal Trauma: Potential complications
- Secondary hemorrhage 10 – 14 days after coming to ER for an injury. May not see hematuria if pt is hypotensive r/t the original injury...they won't be making urine until their fluid levels, BP come up anyway, so you may not even see it right away.
- Paralytic ileus d/t blood into peritoneal space.
- Hypertension
- Renal failure
- Renal atrophy...just a non-fxnl piece of tissue
- Hydronephrosis (d/t a blockage)
- Chronic pylonephritis from scar tissue
- Renal artery stenosis from scar tissue as well

Renal Trauma: Nursing Diagnoses
- Hypovolemia
- Hemorrhage
- Acute pain
- Fear
- Altered mental status
- Altered fluid and electrolytes
- Impaired tissue perfusion

Renal Trauma: Nursing Care
- Bed rest...immobility and all the resulting problems need to be treated
- Broad spectrum antibiotics
- Monitor:
  - for signs of **shock**, infection, abscess, **renal failure** that will develop on that side. Keep other kidney healthy!
  - VS a 1 hr
  - I/O
  - Labs
  - Respiratory status
- Hypertension
- Pain management
- Wound care
- Fluid management. Urine output becomes very important!

Renal Cancer (difficult to diagnose)

Renal Cell Cancer is most common form of cancer to attack the kidneys, with 40,000 cases each year. It accounts for 3% of adult malignancies and rising. It is most common in Northern Europe and North America, with the incidence among African Americans rising. It occurs more often in men than women, and affects people age 40-60 years old.

Renal Cancer: Causes

- Cigarette smoking
- Obesity (the cancer is difficult to find b/c they dont' notice swelling or masses)
- Hypertension (and ironically, tx for HTN)
- Unopposed estrogen therapy (when you have a woman who is going through menopause and requests estrogen therapy, if that woman still has her uterus, you have to treat her with estrogen AND progesterone...if you don't the endometrial lining of the uterus will enlarge (endometrial hypertrophy), and anytime you have LOTS of new cells there is the potential for a cancer cell to develop. The kidneys will ALSO hypertrophy and will make more cells.
- Occupational exposure: petroleum, heavy metals, solvents, asbestos

Renal Cancer: Clinical manifestations

- Hematuria (painless hematuria is cancer until we prove it isn't...it's either bladder cancer or renal cancer)
- Abnormal urine color
- Flank and back pain (chronic and not excruciating and acute like a kidney stone)
- Lump or mass d/t swelling of the kidneys
- Abdominal pain , swelling (gaining weight but don't know why)
- Weight loss (d/t loss of muscle mass)
- Testicle enlargement in men
- Fatigue
- Fever
- Anemia (d/t lack of erythropoietin, and anemia of chronic disease...so two reasons!)
- NOTE: Renal cancer will metastasize to liver, colon and pancreas...bad news. Tissue of origin is the epithelium of the proximal convoluted tubule.

Renal Cancer: Nursing Diagnoses

- Fear
- Ineffective coping
- Knowledge deficit
- Fluid / electrolyte imbalance
- Fatigue
- Pain
- Fear

Renal Cancer: Nursing Care

Need to differentiate between UTI, stones and cyst/cancer.

- Address common side effects of chemotherapy (N.V, fatigue, dizziness, malaise)
- Pace activities to help with fatigue
- Antiemetics for nausea and vomiting
- Dietary consult to encourage nutritional intake (ex: tea time/baked bread on the oncology unit)
- Immunosuppression, neutropenic precautions
- Therapeutic time to aid in expression of concerns and feelings

Polycystic Kidney Disease

- Genetically inherited kidney disease (chromosome 4 and 16), not a cancer but almost treated like a cancer in ways.
- Genetic mutation causing cysts to form in tubules of kidneys...block work of the nephron
- As cysts enlarge, the kidneys functioning units are destroyed
- Will be on dialysis or get a kidney transplant
- Prevalence in US 400,000 with 1800 developing ESRD yearly
- Found throughout world, no preference for race or ethnicity

Polycystic Kidney Disease: Pathophysiology

- Cysts form in renal tubules
- As cysts advance in number and size, the kidneys become grossly enlarged (this large kidney is painful)

- Creates pressure against parenchyma, alters fxn of nephrons
- Interferes with renal filtration and circulation (and solute clearance is compromised)
- Pt accelerates toward renal failure
- Renin release is altered so chronically Hypertensive and not responsive to HTN meds (need aggressive HTN Tx)
- Cysts rupture and bleed, causing hematuria
- Cysts may develop on other organs (adrenalls, spleen, liver)
- Pt is susceptible to urinary obstruction from hemorrhage into cysts.

Polycystic Kidney Disease: Clinical manifestations
- Urine output is usually fine, but Na, an K may be out of whack.
- Hematuria (not painless as in with cancer, there is some back pain)
- Increasing abdominal girth
- Abdominal mass
- Pain at CVA (costo-vertebral angle)
- Proteinuria (d/t glomerular membrane not functioning properly)
- S/S of UTI (burning, frquency, urgency)
- NOTE: may get treated for months and years with abx...until sent to nephrologist.

Polycystic Kidney Disease: Nursing Diagnoses
- Anxiety
- Acute pain
- Fluid and electrolyte imbalance
- Acid / base imbalance
- Knowledge deficit
- Potential infection

Polycystic Kidney Disease: Nursing Care (most of these pts are in hospital b/c getting transplant)
- I/O, VS (watch for HTN)
- Monitor labs
- Pain control
- Patient teaching and emotional support
- Antibiotics
- Avoid NSAIDS (why? NSAIDS interfere with most anti-HTN meds, and will not be cleared well d/t low GFR)
- Monitor CBC, Chemistry
- Adequate fluids

Rhabdomyolysis
- An incredible amount of muscle tissue break-down in which myoglobin is released (you'll see it in urine)
- The myoglobin is hard for the kidneys to handle, and is a major cause of acute renal failure
- Can be caused by a myriad of conditions, usually direct injury to the muscle (big trauma pt)

Rhabdomyolysis: Causes
- Direct muscle injuries
- Drugs: toxins (gangrene also)
- Infections
- Ischemia (if pt lays in one position for 24 hours, the muscles will get ischemic, so someone who passes out or falls and no one notices...this person will get rhabdo)
- Electrolyte imbalances
- Metabolic disturbances
- Severe burns (biggest cause along with crush)
- Crush injuries
- Tetanus

Rhabdomyolysis: Pathophysiology
- Disruption of normal muscle structure
- Lysis of cell membranes releases all content causing myoglobinemia
- Myoglobinemia is filtered by the kidneys causing tubular obstruction (it's a BIG protein molecule)
- Myoglobinuria ensues (urine looks like mud...brown murky color, very thick and almost syrupy)
- Hypovolemia ensues d/t the protein having an oncotic effect of pulling fluid from the vascular and cellular space...an osmotic diuresis effect.

Rhabdomyolysis: Clinical presentation
- Redness / swelling over tender muscles
- Muscle stiffness / weakness
- Muscle paralysis may develop
- Hypotension / hypertension (may be hypotensive d/t dehydration....if early in the process will be hypertensive d/t renin-angio pathway b/c kidneys have sensed low flow of the sludge that is trying to go through there).
- Decreased urine output (will drop off to almost nothing)
- Dark urine with debris and sediment (looks like mud)

Rhabdomyolysis: Nursing Care
- IV fluids...load them with fluids...flood the myoglobin out!
- Monitoring for renal impact (BUN, CR, electrolytes)
- Diuretics (almost a form of dialysis to flood with fluid and give lasix at same time...flushes toxins out)
- Re-establishing fluid balance
- Avoidance of infection r/t injuries, ischemia, tissue damage, stasis of blood
- Meeting metabolic needs
  - Oxygen
  - Nutrition

Lets Review
- List findings in pylonephritis. Flank pain, flu-like symptoms, malaise, leukocytosis, fever, N/V, UTI s/s
- List hallmarks of glomerulonephritis: coca cola urine and severe hypertension
- What are possible causes of rhabdomyolysis? crush injuries, burn injuries
- Define myoglobinuria, why is it a problem?
- What is polycystic kidney disease? cysts blocking tubule, they rupture and bleed, kidneys get enlarged and stop working
- List risk factors for renal cancer: smoking, obesity, unopposed estrogen, toxins,

Renal Failure: Acute, Chronic, ESRD

Acute Renal Failure (often happens d/t medical tx)
ARF is a rapid decline in renal function that can occur in a few hours/day or several weeks. It is relatively asymptomatic (other than urine output dropping off). It is most often detected by labwork. The hallmark findings: decreased u/o, elevated BUN and Cr (may be just BUN in some cases?).

Acute Renal Failure: Causes are many
- Ischemic acute tubular necrosis (causes damage to parenchyma)
- From major burns
- Prolonged surgery
- Sepsis
- Myoglobin
- Nephrotoxic drugs (mycins, NSAIDS...especially in the elderly. NSAIDS cause intrarenal vasoconstriction and potentiate acute tubular necrosis...this is made worse by hypovolemia, chronic renal insufficiency and chemo)
- hypovolemia
- Renal artery stenosis
- Multiple myeloma

- Radiation therapy (causes endothelial swelling)
- Contrast induced nephropathy (see problems 24-48 hours after the dye...most of the time it resolves over about a week in a healthy pt. The dye causes vasoconstriction)
    - Pt at risk for dye-induced nephropathy are those with DM, repeated dye studies and taking nephrotoxic drugs.
- Ex: 35- year old who gets in skiing accident, has a little HTN, but otherwise healthy. Kidneys are impaired by this HTN they have which may or may not be treated, or treated poorly. End up in ER...have a hemorrhage condition, a little hypovolemic and kidneys vasoconstrict...we give fluids and send off to CT scan and give them contrast material...they get contrast-induced nephropathy (CIM)...kidneys impacted by low-flow AND contrast...go into pre-renal failure (means its happening between heart and kidney--the circulation between heart and kidney--, not a "time" thing, but a "position" thing).

Acute Renal Failure: Pathophysiology/ 3 TYPES

- Pre renal (spatial, not time): Decreased blood flow to kidney (a hypovolemic event)
- Intra renal: Damage to renal parenchyma (med or toxin)
- Post renal: Obstruction causing hydronephrosis (kidney stone or tumor)

Acute Renal Failure: Major complications to renal failure

- Hyperkalemia (a major concern...caused by decreased excretion of K and increased cellular release of K through tissue breakdown and acidosis.)
- Hyponatremia (retained fluid causes dilution of urine...but hypernatremia can occur if sodium is unable to be excreted in sufficient amounts...sodium can be variable.
- Hypocalcemia (a result of decreased conversion of Vit D to intrinsic factor needed to absorb Ca from the GI tract...also, Ca is inverse with Ph)
- Hyperphosphatemia (a result of decreased clearance...also inverse with Ca)
- Hyperuricemia (a result of decreased clearance of uric acid)
- Hypermagnesemia (Mg is primarily excreted by the kidneys...can also occur from Mg-containing drugs such as antacids)
- Metabolic acidosis (The kidneys play an important role in maintaining normal pH by urinary excretion of excess hydrogen ions. Metabolic acidosis can result from two problems: First it develops as the result of the kidney's inability to excrete hydrogen ions: as hydrogen ions accumulate, the pH falls and an acidotic state develops. Second, renal failure also can cause metabolic acidosis by loss of renal bicarbonate buffering capabilities. In addition, acidosis potentiates the effects of potassium on the heart, which increases the risk of life threatening dysrhythmias.)

Acute Renal Failure: Phases of renal failure

- There is a beginning, middle and an end
- Onset – initiating phase: basically the precipitating event, 24-48 hours during which tubules become damaged r/t low flow, toxin, etc... Don't see S/S at this point b/c renal injury not yet established, maybe just see changes in the labs. You should be aware of which pts are high-risk for development of ARF...take measures to ensure MAP stays above 70 and that hydration and oxygenation status are optimal. These steps can help prevent further insult to the kidneys.
- Maintenance phase:--A very sudden drop in GFR (about 48 hours after, may last for several weeks...this reflects acute injury to the renal tubules). Protein in urine, glucose in urine...urine output falls...may go from oliguric (400 ml/ 24 hr or less) to anuric (75 ml/24 hrs or less). Pt rapidly develops azotemia (waste products in blood, mainly urea), hyperkalemia, and fluid volume overload.
- Recovery phase: IF we are able to recruit nephrons again, urine output will gradually go up...we will see a normalization of electrolytes...this phase may last for months (slow process). When the tubules are sufficiently healthy, urine output will slowly go up and metabolic waste levels will begin to decrease in the serum. Though urine output is going up, urine concentration issues may continue as the kidneys go through this phase. The kidneys will be extremely vulnerable during this phase, and the pt must avoid the use of nephrotoxic agents or further hypoperfusion states.

Acute Renal Failure: Nursing Diagnoses

- Fluid imbalance
- Electrolyte imbalance
- Risk for infection
- Anxiety
- Impaired tissue perfusion
- Altered mental status
- Knowledge deficit
- Decreased cardiac output

Acute Renal Failure: Nursing care

- Assessment
- History
- Address potential complications
  - Fluid overload (Fluid overload can result in development of congestive heart failure and pulmonary edema. Interventions focus on preventing fluid excess or regaining fluid balance. Fluid restriction, diuretic therapy and dialysis. Watch IV's carefully, administer diuretics as directed and observe for response.)
  - Catabolic processes (breakdown of muscle tissue; hypermetabolic state is triggered by high stress levels, infection, trauma and other acute problems. Hypermetabolic state significantly increases nutritional requirements, particularly protein.)
  - The injured kidneys, however, cannot rid the body of mitrogen waste and the uremic toxins. They accumulate rapidly and produce azotemia. Elevated concentrations of nitrogenous waste products impair the functions of multiple body systems (as we have seen) The brain (renal encephalopathy) and the GI tract (bleeding) are at particular risk for serious complications. Watch weight, serum proteins, mental status .
  - Electrolyte Imbalance
    - Monitor EKG for hyperkalemia...K around 6 shows as tented T-wave...it will grow as the K rises, until the T wave is almost as tall as the QRS...the QRS widens out...the heart will stop beating when the K gets too high.
    - A lot of pts with chronic disease (ESRD) have adapted to K around 7-8...it has gone up slowly. Cation exchange resins may be used either rectally or orally to remove potassium from the body. Sodium bicarbonate, insulin, or hypertonic glucose may be ordered to attempt to drive potassium back into the cells. Dialysis may be ordered to control potassium, particularly when the hyperkalemia is accompanied by excess fluid volume.
    - Sodium levels vary in acute renal failure. Management depends on whether levels are normal , high or low. The close relationship between sodium and water make it important to control; therefore, values are monitored closely. Sodium is restricted in the diet and in IV fluids to control fluid excess and prevent dilutional hyponatremia. Maintaining a balance of intake and output helps prevent or control hypernatremia. If renal function is sufficient, diuretic therapy maybe ordered to lower sodium levels.
  - Acid – base imbalance: Metabolic acidosis: can become severe in acute renal failure, creating disruption of normal cellular functions. The nurse monitors the patient for the clinical manifestations of electrolyte imbalances, particularly potassium, sodium, calcium, phosphate and magnesium. The patient's arterial blood gas values are closely monitored to evaluate acid – base status. Sodium bicarbonate may be ordered to minimize the hypernatrememic effects. Dialysis may also be initiated to help control acidosis. Watch respiratory rate, and cardiac rhythm.
  - Infection: Infection: a major cause of death from arf because of an immunocompromised status. The nurse focuses care on monitoring the patient for signs and symptoms of infection. Scrupulous hygienic maintenance is necessary to minimize the risk of infection. Major sources of infection in the acute renal failure patient include urinary tract infection, pneumonia, septicemia and skin / wound infections. Minimal use of invasive lines and tubes is crucial. Antibiotic therapy requires dose adjustments based on the severity of renal impairment. If antibiotics are ordered, the nurse monitors for the therapeutic and non therapeutic effects of therapy.

Lets review ARF

- Congestive heart failure, hemorrhage and shock are examples of possible etiologic factors for development of which type of renal failure? Pre-renal failure
- What percentage of critically ill patients develop ARF? 10-25% (higher in ICU sometimes)
- Acute Tubular Necrosis is caused by what? toxins (-mycins, contrast, lasix can also be toxic as well, so push slowly)
- Renal tissue ischemia occurs when the MAP drops to less than 60.
- Rhabdomyolysis results from which type of tissue breakdown? Skeletal muscle
- Agents that are considered highly nephrotoxic include>>> aminoglycocides (-mycins), dyes, NSAIDS
- When urine output falls below what ml per 24 hours the term oliguria applies? 400 ml
- What percent of nephrons may be lost before significant renal dysfunction is noted? 50% (also saw 80%)
- The onset phase of acute renal failure is associated with what manifestations? not much, just lab changes
- The maintenance phase ends when the BUN and Cr? start to go back to normal.
- Intrinsic Renal Failure is caused by ? damage to kidneys from HTN, DM, nephrotoxic agents, ATN.
- What type of electrolyte imbalances most commonly occur secondary to ARF? hyperK and hypoNa, hypoCa
- Becomes acidotic
- Hyperkalemia is associated with what problems? cardiac dysrhythmias (conduction issues and contractility issues)

DIALYSIS

Hemodyalisis

Hemodialysis is most common...diffuse particles across a membrane from one fluid compartment to another to take out excess K, Ph, H ions, etc..it does not fix the kidney. It just fixes the metabolic problem associated with the kidney not working...helps to normalize electrolyte and acid/base

Hemodialysis Indications

- Used in
  - Acute poisoning (Tylenol or barbituates)
  - AFR, CRF
  - Transfusion reaction (dialyse out the destroyed RBCs d/t transfusion)
- Disadvantages
  - Vascular access (always a source of infection, sometimes work/sometimes don't)
  - Restricts activity (can't play sports with a shunt!)
- Contraindications (when we CAN'T do dialysis)
  - Coagulopathy, hemodynamic instability (dialysis will drop the pt's blood pressure b/c a good volume of the blood will be in the machine at any given time)

Hemodialysis: How it Works

- Cleans the blood by pumping it out of the patient via a venous access
- Passes through a dialyzer
- Removes fluid and solutes
- Returns the filtered blood back to the patient via artery (don't want it to clot in the process!)
- Takes about 5-6 hours

Hemodialysis: Complications

- Infection
- Decreased cardiac output
- Cardiac arrhythmias
- Disequilibrium syndrome
- Altered mentation
- Air embolism
- Disconnection hemorrhage (someone trips over tube...uh-oh!)

Continuous Renal Replacement Therapy (CRRT)
This is a new type of dialysis that is used when hemodialysis is not feasible (i.e hemodynamic instability). It is primarily seen in the critical care setting (Used in ICU, pt is very sick). Frequent assessments and ongoing monitoring is essential! It is a continuous form of therapy, so it's more slow and not as much blood removed at any one time (10-24 hr project)

CRRT: Indications:
- Multiple organ dysfunction syndrome (d/t sepsis)
- Sepsis
- Acute renal failure
- Inability to tolerate hemodialysis
- CRRT

CRRT: Disadvantages
- Requires vascular access
- Slow process
- Restricts activity level (can't get up and do PT!)
- Risk of contamination

CRRT: Contraindications
- Acute poisoning (too slow for this)
- Hct > 45%
- Inability to anticoagulate (maybe pt had a AAA repair, so you can't give them anticoagulant)
- Low MAP (< 50)
- CHF because of very low diffusion state (we do dump fluid into the system with CRRT)

CRRT: Variations
- Continuous arteriovenous hemofiltration (CAVH)
- Continuous arteriovenous hemofiltration –D (CAVH-D)
- Continuous venovenous hemofiltration Dialysi (CVVH-D)

Peritoneal Dialysis
- Uses the peritoneal lining to serve as the semi permeable membrane
- Diffusion, osmosis and filtration occur
- Removes metabolic wastes and correct fluid and electrolyte imbalances
- Fluid is introduced into peritoneal cavity via a peritoneal catheter, we allow it to sit and then we dump it out. The peritoneal membrane exchanges electrolytes back and forth...helps to normalize Na, K, etc... Can correct some fluid and electrolyte imbalances
- Trocar introduced into peritoneal space...2 L at a time (warmed)...let it sit for 25 minutes, then drain it out (turn pt a few times)

Peritoneal Dialysis: Indications for use
- Hemodynamic instability
- A way to dialyze people at home...many people do it at night.
- Severe cardiovascular disease
- Hemodialysis not available (live in rural area)
- Less rapid treatment is appropriate
- Inadequate vascular access (veins too fragile)

Peritoneal Dialysis: Disadvantages
- Slower
- Abdominal discomfort (especially if fluid is cold...so warm the fluid)
- Decreased mobility

Peritoneal Dialysis: Contraindications
- Adhesions of peritoneum
- Peritonitis
- Recent abdominal surgery

Peritoneal Dialysis: Complications
- Infection is a big deal...so you inspect the return. If cloudy send off for a culture.
- Fluid overload
- Hyperglycemia
- Respiratory insufficiency
- Metabolic alkalosis
- Abd pain

Drugs and Dialysis
- There are drugs that can not be cleared by the kidneys
- During dialysis drugs are removed form the blood and brought down to sub-therapeutic drug levels
- Anti-hypertensives and nitrites should not be given prior to dialysis (would exacerbate hypotension)
- Some drugs dialyzed out of blood, some drugs not dialyzed out of blood...so give ABX after dialysis so the abx stay in the system and do their job (may give before and after, ask MD).
- Dialyzability of common drugs (see slide for this..there's more here that didn't copy over)
  - Dialyzed
    - Beta blockers
    - Penicillin drugs
    - Amonoglysides
    - Cephalosporins
    - Acetaminophen
    - ASA
    - Captopril
  - Non-dialyzed drugs///be careful for toxicity...they will stay in the system longer than in a healthy pt.
    - Albumen
    - Diazepam
    - Digoxin
    - Furosemide
    - Heparin
    - Iron products
    - Levothyroxine
    - Nifedipine

Chronic Renal Failure: High Acuity Patient
- Slow progressive, irreversible destruction of the kidney
- Most common causes = ARF that was not reversed, DM and HTN
- 375,000 patients in US with ESRD
- 5 year survival rate is 35%
- If diabetic 5 year survival rate is 20%
- > 275,000 people on dialysis
- 100,000 with functioning renal transplants

Chronic Renal Failure: Stages
- Diminished renal reserve: lasts until GFR drops to about 50% of normal. Nephrons are being destroyed byt adequate compensatory mechanisms are in place to maintain normal renal function. Hypertrophy of remaining nephrons increases their filtration capacity. The person is clinically unaware of this stage because of lack of clinical manifestations. Nephrotoxins can cause further renal insult.
- Renal insufficiency (CR approaching 2.0-2.5): begins when the GFR has dropped to 20 – 50% of normal. Heralded by development of polyuria that is the same SpG of plasma. The kidneys have lost the capacity for concentrating

the urine. There is loss of sodium and water. Any additional stress placed on the remaining functional nephrons can result in rapid progression to renal failure.
- Renal failure CR approaching 3.0: begins when the GFR has dropped below 20% of normal. Volume and solute regulation is lost and the person develops metabolic acidosis, hyperkalemia, and edema. It is at this point that the person may develop signs and symptoms of uremia, which are multisystem in nature.
- End Stage Renal Disease, anuric, GFR is < 5% of what it should be....kidneys are abnormal looking and nonfunctional. requires dialysis. Structurally, the nephrons are scarred with loss of capillaries and tubular fibrosis and atrophy. Kidneys are grossly abnormal, shriveled in appearance and non functional. Dialysis or renal transplantation is required. Uremia is the primary characteristic of this stage.

Uremic Syndrome
- Constellation of clinical findings due to decline in renal function
  - Fluid overload
  - Electrolyte abnormalities (often live with K of 7-8)
  - Carbohydrate intolerance (so glucose is always high)
  - Abnormal protein metabolism (muscle wasting...thin extremities, very weak)
  - Increased lipid (LDL is high)
  - CNS changes (poor information processing, headache d/t hyponatremia, don't sleep well, restless leg syndrome, parasthesias)
  - M/S: Bones become brittle and break, they hurt (osteodystrophy) this is d/t loss of Ca
  - CV: HTN, edema, CAD, dysrhthmias, pericarditis, effusions, heart failure (heart becomes fluid overloaded)
  - Congestion in lungs...pleuritis, pulmonary edema, often have Kussmaul respirations d/t sugar being high and acidotic...trying to blow off CO2
  - GI motility decreased, anorexic, N/V, gastroparesis
  - Endocrine: hyper-parathyroid (trying to increase Ca levels), glucose intolerance
  - Feet and legs get uremic frost...paresthesias, tingling, neuropathies, infections, dry skin, itching, stress Fx)
  - Hematologic: anemia, impaired clotting
  - Reproduction: amennorhea and impotence
  - Immune: diminished leukocyte count, increased susceptibility
  - These pts border on CHF...they have elevated K, glucose and lipids

Chronic Renal Failure: Nursing Diagnoses
- Altered fluid and electrolyte balance
- Altered nutrition: less than
- Activity intolerance
- Risk for infection (very immunocompromised)
- Impaired tissue perfusion
- Impaired skin integrity (prone to breakdown from uremia)
- Impaired oxygenation (if renal failure and COPD then BIG problems...not a good combo to have)
- Risk for injury/impaired mobility (break easily)

Chronic Renal Failure: Patient goals
- Preserve renal function
- Postpone need for dialysis or transplant as long as possible
- Improve body chemistries via fluid and diet
- Reverse organ system alteration (fluids, lasix)...not totally reversible, but can re-recruit some nephrons
- Provide comfort and improve quality
- Prevent injury and infection

Chronic Renal Failure: Foods high in potassium AVOID THESE!!!
- Oranges/ juice bananas tomatoes greens salt substitutes nuts
- Cantaloupe peaches tomato sauce potatoes avocados
- asparagus coffee/tea chocolate milk products bran

Chronic Renal Failure: Nursing Care

- Dialysis is required...help them cope with this and plan their life around it.
- Chronic anemia, hypoxia
- Increased risk of infection (lots of teaching)
- Immunosupression (lots of teaching)

Chronic Renal Failure: Nursing Care (cont'd)

- Monitoring of all major body systems
- Rapid recognition of development of complications
- Timely interventions

Lets Review

- The inability of the kidneys to produce the active component of vitamin D lowers which electrolyte? Ca
- Metabolic acidosis is closely associated with acute renal failure because of the kidney's inability to? get rid of H ions...causes problems such as CNS depression, tachypnea, Kussmaul respirations
- The BUN/ Cr ratio is considered a good indicator of probable ARF when it rises to: Normal is 20:1...30:1 is pre-renal, 10:1 is intra-renal
- Major complications that are routinely addressed in the patient with ARF include: hyperkalemia, metabolic acidosis and catabolism, fluid overload often, infection.
- What foods containing high potassium contents should be avoided in renal failure? orange juice and bananas, tomatoes
- Hyperkalemia is frequently treated with what pharmacologic agent? Kayexalate (binds up K...can be given enema or oral) Also an issue of high potassium in cellular space as well as vascular space, so that's why we don't go the insulin/ glucose route (also pt does not utilize insulin well and is already hyperglycemic)
- The major purpose of using dialysis is to? correcting imbalances in fluid and electrolyte, getting rid of metabolic waste.
- When peritoneal dialysis is performed, the semipermeable membrane is the: peritoneal membrane
- List three major causes of chronic renal failure: DM, HTN, ARF untreated
- The renal failure stage of chronic renal failure begins when the GFR has dropped to what percent of normal? < 20% ??

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