Etiology of ARF

Three categories of causes: Prerenal (inadequate renal perfusion), Intrinsic (diseases within renal parenchyma), and Postrenal (obstruction). More specifically:

Prerenal: Decreased effective intravascular volume leading to total body volume depletion. Examples are hemorrhage, GI loss (vomiting, diarrhea), renal loss (diuretics, salt-losing nephritis), skin loss (burns, sweating). Also, volume redistribution such as third spacing of fluid (peritonitis, ascitis, pancreatitis, hypoalbuminemia) or increased vascular capacity (sepsis, anaphylaxis). Decreased cardiac output from left-sided failure (CHF, cardiomyopathy, and pulmonary embolism), right-sided failure, valvular heart disease, or pericarditis, tamponade. Renal vascular disease. Severe dehydration, circulatory collapse; electrolyte imbalances (hypokalemia, hyponatremia).

Intrinsic: Glomerular disease: Most of the glomerular disease producing ARF are felt to be immunologically mediated and result in disrupted glomerular filtration. Progression of the inflammatory process leads to destruction of glomerulus and a decrease in the GFR. Primary disease from post-streptococcal infection, rapidly progressive glomerulonephritis or secondary to systemic disease (SLE, vasculitis, endocarditis). Tubulointerstitial disease such as ischemic acute tubular necrosis from trauma, surgery, or septicemia, nephrotoxin (aminoglycoside, radiocontrast, pigment, CCl_4, methoxyflurane, antibiotics), allergic interstitial nephritis (drug-induced), interstitial (non-drug) nephritis (immune complex), metabolic toxin (uric acid, Ca++), or heavy metals (cis-Platinum). Vascular such as atheroembolic or small vessel (scleroderma, malignant HTN, DIC, TTP, bilateral renal infarction)

Postrenal: A simple mechanical or functional obstruction to the free flow of urine preventing its excretion, thereby producing renal failure. Can be caused by bladder neck obstruction d/t prostatic disease or denervation. Can be elevated by suprapubic palpation, percussion for enlarged bladder, or postvoid bladder catherterization to measure residual volume. Bilateral urinary tract obstruction causes include: retroperitoneal fibrosis, tumors, abscess, surgical accident, or from stones, papillary tissue, blood clots, or pus causing obstruction. Intrarenal obstruction is usually d/t intratubular precipitation of poorly soluble material such as: uric acid (tumor chemo), oxalic acid (ethylene glycol overdose, methoxyflurane anesthesia, small-bowel bypass surgery), methotrexate (insoluble metabolites), acyclovir, sulfonamides (out-dates, insoluble-compounds), or myeloma proteins.

Other causes: NSAIDS, ACE inhibitors (decrease glomerular capillary perfusion pressure). These agents can cause ARF in those w/ bilateral renal artery stenosis, unilateral renal artery stenosis without a contralateral kidney, or other high-renin disorder states. Commonly used therapeutic agents that can induce ARF d/t acute interstitial nephritis include: furosemide, penicillin, phenytoin, sulfonamides, rifampin, trimethoprim, cimetidine, and captopril. Rhabdomyolysis and Myoglobinuria ??? Nephrotic syndrome can lead to ARF. Disorders of the blood, such as idiopathic thrombocytopenic purpura (ITP), transfusion reaction, or other hemolytic disorders. Causes also include malignant hypertension and disorders associated with childbirth such as postpartum renal failure and bleeding associated with placenta abruptio or placenta previa. Autoimmune disorders such as scleroderma may cause acute renal failure; hypercalcemia may cause ARF but also may result from renal failure

Etiology of CRF

An irreversible deterioration of renal function leading to the clinical syndrome of uremia. The definition of CRF requires that at least 3 months of renal failure have occurred

Causes: Congenital (polycystic kidney disease, Alport's syndrome, congenital hypoplasia), Glomerular diseases (proliferative GN, membranous GN, crescentic GN (RPGN), glomerulosclerosis - focal & segmental), Secondary GN (DM, amyloidosis, SLE, PAN), Interstitial diseases (chronic pyelonephritis, VU reflux, analgesic and reflux nephropathy, TB, nephrocalcinosis, schistosomiasis), Vascular diseases (arteriosclerosis - commonly due to HTN, Vasculitides (PAN, SLE, scleroderma), obstructive uropathy (stones, strictures, retroperitoneal fibrosis, prostatic hypertrophy, pelvic tumors). Also, repeated infections and scarring result in bilateral small scarred kidneys, progressive loss of renal function - sometimes occurs even if the underlying condition is corrected, and "Uremic syndrome" may be caused by the accumulation of not only urea, but also phosphate, parathyroid hormone, creatinine, phenols, indoles, and guanidine. HTN alone is also a cause. Progressive over a long time and may go unnoticed until its too late! Episodes of ARF (usually acute tubular necrosis) often lead, eventually, to CRF;

How ARF is Diagnosed

Increased serum BUN (azotemia with isosthenuria or minimally concentrated urine), serum creatinine, and 25-OH Vitamin D; hyperkalemia; hyperphosphatemia; metabolic acidosis; ½ of all patients nonoliguric (therefore cannot be used as single diagnostic); renal biopsy, rectal and vaginal examinations and attempts at bladder catheterization are done when obstructive uropathy is suspected, urinary sediment may show WBCs, RBCs, and casts (granular and tubular cells), urinary eosinophils suggest an allergic tubulointerstitial nephritis; RBC casts suggest glomerulonephritis or vasculitis, X-ray of the abdomen can detect 90% of urinary calculi that are radiopaque, ultrasonography to detect stones; if obstruction is strongly suspected, antegrade or retrograde contrast studies can establish the site of obstruction and suggest therapy. A postvoiding urethral catheterization assesses bladder outlet obstruction; Kidney or abdominal ultrasound is usually the best test, but abdominal X-ray, abdominal CT scan, or abdominal MRI may also reveal the cause of the acute renal failure. Kidney size is usually normal or slightly large. Chemical tests of blood and urine (such as fractional excretion of sodium or fractional excretion of urea) may help to distinguish the causes. A clean catch urine specimen will indicate if the cause is infection within the urinary tract. Renal angiography (renal arteriography) may be used to diagnose causes within the blood vessels of the kidney; Urine/Serum Creatinine Ratio, Renal Failure Index, Urine/Serum Urea Ratio, and Free Water Clearance can all be used; normochromic-normocytic anemia; hyponatremia (fluid overload); edema; Urine sodium concentration is usually greater than 40 mEq/liter and urine osmolality is low because of abnormalities in tubular function; Hyaline, granular, and pigmented tubular casts, tubular dilatation, tubular regeneration, interstitial edema; evidence of CHF; loss of skin turgor; orthostatic hypotension and tachycardia; elevated postvoiding residual volume; decreased GFR; if ATN, may have smoky urine with dark granular casts (20% may be normal); in

glomerulonephritis, hematuria and proteinuria will be present; secondary liver failure(increased SGOT, SGPT);BUN elevated out of proportion to serum creatinine, often greater than 20:1; low urine Na+.

How CRF is Diagnosed

Blood pressure may be high, with mild to severe hypertension. A neurologic examination may show polyneuropathy; abnormal heart or lung sounds may be heard; uremia present; abnormal urinalysis; increased BUN, creatinine; decreased creatinine clearance; decreased GFR; hyponatremia; renal or abdominal X-ray, abdominal CT scan, abdominal MRI, abdominal ultrasound can help distinguish cause; increased PTH; renogram; increased Mg++ levels; increase in hyaline casts; white nail syndrome; low urine osmolality; low urine Na+; complete blood count (anemia can occur, low EPO levels); consider complement levels, protein electrophoresis, antinuclear antibodies, ANCA; renal biopsy; bone evaluation (DEXA test ??); Plasma Na+ concentrations may be normal or reduced. The serum K is normal or only moderately elevated unless K-sparing diuretics, ACE inhibitors, beta-blockers, or angiotensin receptor blockers are taken. Abnormalities of Ca, phosphorus, parathyroid hormone (PTH), vitamin D metabolism, and renal osteodystrophy can occur; hypocalcemia and hyperphosphatemia are found regularly; moderate acidosis; anemia; kidneys reduced in size; irregular radiolucencies seen radiographically; high carbamylated Hb; hyperkalemia; hyperphosphatemia; metabolic acidosis.

Physiological Effects of ARF

Oliguria; anuria; dark urine; reduced GFR; azotemia; edema; inflamed interstitial tissue; hypocalcemia, hyperphosphatemia, secondary hyperparathyroidism d/t temporary loss of calcitriol production by the injured kidney and phosphate retention. Hypocalcemia may be profound in patients with myoglobinuric ARF, apparently due to the combined effects of Ca++ deposition in necrotic muscle, reduced calcitriol production, and resistance of bone to parathyroid hormone (PTH); hypernatremia; hyperkalemia; acidosis; gout; fluid retention; kidney stones; chronic interstitial nephritis; excretion of glucose, a. acids, P, and bicarbonate in the urine w/Fanconi syndrome; nephrocalcinosis; pyelonephritis; hypercalciuria; calcium oxalate stones; struvite stones; tissue breakdown; uremia; hematuria and HTN w/nephritic syndrome; normochromic-normocytic anemia; hypovolemia; weight gain from water; simultaneous CHF, DM; HTN; decreased T4; decreased serum Cl w/ diuretics and bicarbonate; decreased urine specific gravity (inability to reabsorb water); low urine Na+; depressed EPO synthesis; increased RT3U; neuropathy; elevated ESR; decreased urine amylase; increased SGOT; increased 25-OH vitamin D; auscultation of heart and lungs from fluid retention; N/V; seizures; vomiting blood; fatigue; change in mental status; depressed albumin and prealbumin; dehydration and ECF depletion in patients requiring cholecystography or renal insufficiency requiring urography may occur; hypokalemia, hyponatremia, hypomagnesemia, or marked contraction of ECF volume with peripheral vascular collapse can occur; abdominal pain may be seen with anything that causes renal swelling (leptospirosis, pyelonephritis, toxins).

Physiological Effects of CRF

Renal osteodystrophy (osteomalacia from demineralization, osteitis fibrosa cystica from hyperparathyroidism, metastic calcification of joints and soft tissues); increased plasma P levels; decreased serum Ca++ d/t decreased 1,25-Vit.D and increased P; PTH acts on kidney to increase P secretion while retaining extra Ca++, but w/inability to produce 1,25-Vit.D, Ca++ levels must be maintained through bone resorption and P levels low d/t inability to increase gut reabsorption of Ca++; increased serum Ca++; increased serum F; hypoproliferative normochromic normocytic anemia; increased circulating retinol-binding protein; small kidneys; hyperglycemia; hypoglycemia; elevated TG level; atherosclerotic cardiovascular disease (d/t underlying diseases often seen: DM, HTN, nephrotic syndrome); increased VLDL; low plasma-free carnitine levels; protein catabolism; hypophosphatemia w/TPN; postdialysis hypoglycemia; DM pts. may have increased risk of hyperkalemia, retinopathy, neuropathy, gastroparesis, and amputation; proteinuria, hematuria, pyuria; hyponatremia & hyperkalemia; acidosis; increased uric acid levels; Pericarditis; decreased platelet and function of; WBC (decreased HMI & CMI); reduced calcitriol formation; impaired intestinal absorption; reduced hemopoiesis due to toxic effect of urea on BM; loss of EPO production - this may be normal - which is less for the anemia. More EPO in Polycystic disease; reduced life span of RBCs; increased blood loss d/t capillary fragility & general bleeding tendency; amenorrhea (in women); skin pigmentation; hyperprolactinemia (may lead to libido loss); urinary tract infections, kidney stones, obstructions of the urinary tract, glomerulonephritis, and other disorders should be treated as appropriate; decreased GFR; increased Mg++ levels; excessive thirst; increased or decreased urine output; decreased Hb and Hct; parathyroid gland hyperplasia; albuminuria (decreased albumin); depressed albumin and prealbumin; nocturia; Cardiomyopathy; ketosis; hypercholesterolemia (rare); calcified glomeruli and tubules; hyperkalemia; urine specific gravity remains between 1.008 and 1.012 and does not change with variations in water intake; thrombocytopenia; ulcerative stomatitis (ulceration in the oral cavity), erosive gastritis, and uremic colitis may cause gastrointestinal bleeding; fibrinous pericarditis; pancreatitis (rare); calcification and oxalate deposition may also occur in the heart; uremia.

Nutritional Implications of ARF and CRF

Severe constipation w/phosphate binders; N/V; loss of water soluble vitamins (except B12) especially w/dialysis; more prone to infections (decreased WBC); uremia; anemia d/t reduced EPO production and intake of iron & vitamins; malnutrition; edema; amenorrhea in women increased bone loss; unintentional weight loss; refeeding complications w/TPN: hyperglycemia, rebound hypoglycemia, hyper/hypokalemia, hypophosphatemia, hypomagnesemia, hypocalcemia, refeeding edema, diarrhea, N/V (can also be seen with pt's not on TPN); fever (increases nutrient needs); gout (can lead to pain and immobility); decreased albumin can exacerbate edema; risk of bleeding; protein catabolism; increased risk of CVD; hyper/hypoglycemia; stomatitis, and an unpleasant taste in the mouth; metabolic encephalopathy; CHF; abnormal bone

mineralization resulting from hyperparathyroid function, calcitriol deficiency, elevated serum phosphorus, or low or normal serum Ca++ (bone loss); osteomalacia; hypertriglyceridemia; iron, folate, B12 deficiency; ketosis; hypercholesterolemia (rare); HTN; hyperkalemia; secondary hyperparathyroidism; hypocalcemia; acidosis; lack of hunger prior to dialysis; Dialysis pt's may have: hyper/hyponatremia, hyper/hypokalemia, changes in Cl, CO2, creatinine, hyper/hypoglycemia, hyper/hypocalcemia, hyper/hypophosphatemia, high/low BUN, increased uric acid levels, changes in Alk Phos, LDH, serum ferritin, SGOT, high/low cholesterol, low total protein, changes in albumin and low Hct. See Nutritional Treatment (Things I may have missed) for treatment; loss of B-Vitamins w/dialysis; thrombocytopenia; ulcerative stomatitis (prevent NL food intake), erosive gastritis, and uremic colitis may cause gastrointestinal bleeding (exasperate anemia); impaired conversion of Vitamin D to its active form in the kidney.

Lab Alterations Seen in Disease State

Lab Test: Uric Acid
Normal value: 4.1 to 8.8 mg/dL
Alteration: increase
Cause of Alteration: Measures amt. of uric acid in blood. Uric acid is the end product of purine metabolism. The kidneys excrete most uric acid produced in the body. Build-up occurs w/abnormal kidney function. Increased levels of uric acid can cause gout.

Lab Test: Creatinine Clearance
Normal value: male: 97 to 137 ml/min; female: 88 to 128 ml/min
Alteration: decrease
Cause of Alteration: The creatinine clearance is a measure of the glomerular filtration rate, that is, the volume of filtrate made by the kidneys per minute. Creatinine is used for this purpose since it is normally present in the body, and very little creatinine is reabsorbed after it is filtered. The amount of filtrate made in the kidney depends on the amount of blood that passes through the glomeruli and on the ability of the glomeruli to act as filters. Thus, abrupt cessation of glomerular filtration causes the serum creatinine to rise by 1-3 mg/dl daily, which will lead to exiting creatinine to decrease.

Lab Test: BUN
Normal value: 8-25 mg/dL
Alteration: increase
Cause of Alteration: When kidneys are unable to excrete urea normally, it builds up in blood. An increase in BUN is referred to as azotemia.

Lab Test: Serum Creatinine
Normal value: 0.7-1.5 mg/dL
Alteration: increase

Cause of Alteration: Creatinine is excreted from the body entirely by the kidneys. With normal renal excretory function, the serum creatinine level should remain constant and normal. Abnormal kidney function will lead to a rise of creatinine in the blood.

Lab Test: 25-OH vitamin D
Normal value: 16.0 to 74.0 ng/ml
Alteration: increase
Cause of Alteration: Since hydroxylation of 25-hydroxy vitamin D to 1,25-dihydroxy vitamin D takes place in the kidneys, the accumulation of 25-hydroxy vitamin D is an indication of kidney disease.

Medications Commonly Used in Disease State

Medication: Sodium polystyrene sulfonate (Kayexalate)
Action: Antihyperkalemic agent
Nutritional Concerns: Avoid K+ suppl; take Ca++ and Mg++ suppl separately several hours later; anorexia; 33 mg Na+/g drug-500 mg Na+/dose (consider Na+ with low Na+ diet); N/V; constipation; fecal impaction; diarrhea; edema; Na+ retention; hypokalemia; hypocalcemia; confusion; weakness; irritability; irregular heartbeat. Monitor electrolytes, Ca++ and Mg++.

Medication: Furosemide (Lasix)
Action: diuretic (loop, K+-depleting)
Nutritional Concerns: Increase K+, Mg++ (food or suppl); decreased kcal + Na+ may be recommended; Avoid natural licorice; anorexia; increased thirst; oral irritation; stomach cramps; N/V; diarrhea; constipation; monitor rxn to sorbitol in sol'n; limit alcohol; hyperglycemia; possible hypotension; dehydration; rare gout; depressed K+, Mg++, Na+, Cl, Ca in blood/serum; increased glucose, BUN, uric acid, chol, LDL, VLDL, TG in blood; anemia; dyscasias; increased urinary excretion K+, Na+, Cl, Mg++, Ca++, water, & glucose. Monitor BP, electrolytes, Mg++, Ca++, glucose, uric acid, CO2, and renal function.

Medication: Sodium Bicarbonate
Action: Antacid (alkalinizing agent to increase pH)
Nutritional Concerns: Consider Na+ content if Na+ restricted diet; take Fe suppl separately; increase thirst; edema; gastric distention; cramps; gas; caution w/CRF, CHF, HTN; peripheral edema; increase in blood/serum Na+ and pH (IV); decreased blood/serum K+ & Ca++; increased urinary pH.

Medication: Bumetanide (Bumex)
Action: Diuretic (loop, K+-depleting)
Nutritional Concerns: May need to increase K+, Mg++ in diet and decrease Na+ and kcals; caution w/alcohol and decreased renal function; possible hypotension, dizziness, headache; rare gout; depressed Na+, Cl, K+, Mg++, Ca++ in blood/serum; increased

creat, BUN, uric acid, LDH, SGOT, SGPT, alk phos, bil, chol in blood/serum; increased urinary excretion of Na+, Cl, K+, Mg++, Ca++, water, P and decreased uric acid excretion. Monitor BP, electrolytes, Mg++, uric acid, renal and hepatic function, CBC, and CO2.

Medication: Calcium acetate (Phos-Lo)
Action: Phosphate binder
Nutritional Concerns: Avoid Ca++ supplement; decreased Fe absorption; anorexia; N/V; constipation; kidney stones; increased Ca++ in blood/serum; decreases P in CRF. Monitor serum Ca++ and P.

Medical Treatment of Disease
Above drugs, immunosuppressive therapy: cyclosporin, azathioprine, mycophenolate mofetil, FK506, OKT3 and corticosteroid drugs w/transplant. P binders if hyperphosphatemia.

Surgical Treatment of Disease
Hemodialysis (Cannula, AV, artificial loop graft, subclavian catheter)
Peritoneal dialysis (CCPD, CAPD)
Kidney transplant
Parathyroidectomy (Pt's w/secondary hyperparathyroidism affecting bone severely)

Nutritional Treatment of ARF
Pre-dialysis
0.6-0.8 g/kg/day protein for nonhypercatabolic , nondialyzed patient w/ GFR <10 mL/min.(>60% HBV). Those w/nephrotic syndrome (0.8-1.0 g/day)
Energy usually 35 kcals/kg/day (can be as high as 45-55 kcal/kg/day)
Na+ usually 2-4 g/day (depends on disease etiology and urine output)
K+ usually not restricted unless GFR<10 mL/min
May need to limit fruits and increase fat if K+ must be restricted. Limit saturated fats and increase amt. of mono/polyunsaturated fats (P:S:M=10%:7%:13%)
P intake 10-12 mg/g dietary protein
Ca++ should be 1-1.5 g/day
Serum electrolytes must be monitored closely
Oliguric patients (<500 mL urine/day) may have elevated serum K+ & P and decreased Na+
Fluid unrestricted until urine output decreases or edema present
Daily fluid intake for oliguric patients should= urine output + ~500mL for insensible losses. Increased if fever present.
Daily RDA of B-complex, C, D, iron, Ca++, Zn++ may be needed. Individualize as necessary

Hemodialysis

1.2-1.4 g/kg/day protein if hemodialysis (>60%HBV). Diabetic on hemodialysis may need 1.2-1.5 g/kg protein per day.
Calories should be 30-35 kcal/kg
Na+ intake is usually 2g/day
K+ intake usually 2-3 g/day
Not that oliguric and anuric patients may need Na+ restriction of 2-3 g/day and K+ restriction of 1.5-3 g/day
P ~12-15 mg/g dietary protein
1-1.5 g/day Ca++ may be required
Fluid should= output + 1000 mL/day (most anuric patients can handle this)
Vit. C=60-100 mg/d, B6=5-10 mg/day, Folic acid=0.8-1.0 mg/day
Daily RDA for Fe, vit. D, Ca++, and Zn++ may be required. Individualize as necessary
Pt. may require TPN b/c lack of appetite after dialysis. Monitor for fluid overload, edema, and hyponatremia
CAVH or CAVHD may help to avoid the above by removing waste products and maintaining NL Na+, albumin, and water balance.

Peritoneal Dialysis

1.2-1.5 g/kg protein for patients on peritoneal dialysis (>60% HBV)
25-35 kcal/kg including dialysate calories. 20-25 kcal/kg if weight loss desired.
Na+ usually ~2-4 g/day
K+ may be as high as 3-4 g/day
P ~12-15 mg/g dietary protein
1-1.5g/day Ca++ may be required
Fluid should be monitored. Most can tolerate 2000 mL/day
Vitamin/minerals requirements same as hemodialysis. Required to replace losses through dialysis, and b/c lack of albumin. Also, as disease worsens lose 1-alpha-hydoxylase activity thereby lowering active form of vit. D. This will lead to increased PTH levels and bone resorption.

Nutritional Treatment of CRF

Predialysis

0.6 g/kg/day of protein is generally used for protein needs to avoid nitrogenous waste build up in the blood. ~65% should be of HBV
0.8 g/kg/day of protein if patient is malnourished
If patients exhibit significant proteinuria (as in diabetic nephropathy)-then daily urinary protein losses should be added to calculated allowance. Also, corticosteroid therapy and recent surgery may cause protein intake to be increased d/t catabolic state.
35-50 kcals/kg/day to maintain body weight and effective protein utilization
Complex and simple CHO should be given to prevent weight-loss (hard candy, soda, fruit juices, ices, and whole fruits, sugar, honey, and jelly are all ok). If patient on K+ restricted diet then fruits must be boiled to mush allowing for K+ leaching. Also, watch all fruit products.
Increase MUFA and PUFA and limit SFA in diet. Monitor lipid levels in those whose are elevated or at risk

Na+ restriction may be needed to prevent Na+ retention, generalized edema, HTN, and CHF. Restriction can be often be met by 2-3 g Na+/day. Some may need a restriction of 1 g Na+/day
K+ not usually restricted until later stages of CRF. K+ >5.0 mEq/L may require 2-3 g K+/day restriction to prevent hyperkalemia.
Those on ACE inhibitors may need mild restriction of K+
Restriction of dietary P may help to prevent secondary hyperPTism. When GFR <25 mL/min-Ca++ supplementation and P restriction is often required d/t poor Ca++ absorption and lack of P excretion. P restriction at this pt 8-12 mg/kg/day recommended. Ca++ supplements required b/c foods high in P are often good sources of Ca++ and are restricted. Phosphate binders should be given when serum P levels less than 6.0 mg/dL
No fluid restriction until input>output. If edema present may prescribe loop diuretic to increase Na+ and water loss. In late stages of CRF fluid limit is: volume of output + 500 mL/day for insensible losses (prevent edema and hyponatremia)
Vitamin/Mineral supplements usually needed d/t dietary restrictions:
Folic acid=1 mg/day; B6=5 mg/day; DRI for other B-complexes; C=60-100 mg/day; Vit D may be needed; Vit A should not be supplemented (may accumulate and is toxic in large amounts); EPO and Fe supplementation
Renal vit/min supplements also available

Hemodialysis (HD)

Protein intake must at least meet minimum dietary allotment and the maximal allowable amt of protein must not worsen uremia.
Protein needs are same as those on HD w/ARF
Energy needed to maintain or achieve IBW. 30-35 kcals/kg. May need 35-50 kcals/kg for repletion
CRF patients have increased risk hyperlipidemia (elevated LDL and TG and NL to low HDL). Fat should be kept at 30% if pt eating well-may need to increase if not. Lipid lowering agents (statins) may be needed. Avoid fibric acid derivatives (clofibrate, gemfibrozil) because of increased risk of rhabdomyolysis, especially if taken with statin drugs.
Excessive Na+ may precipitate pulmonary edema and CHF. Determine pt BP, BW, and kidney function. Na+ restriction of 2-3 g Na+/day usually prescribed.
Fluid usually 1 L/day plus output if any
K+ intake is individualized. Intake of 1.5-3 g K+/day usually fine
We want to maintain NL serum P level of 4.0-6.0 mg/dL, which will require restriction in foods high in P (meats and milk products) leading to dietary protein restriction
12-17 mg P/kg/day recommended. P binders may be prescribed to increase P excretion. Al+++ containing binders used initially until Ca++ x P product </ 70 or serum P </ 6.0 mg/dL. Calcium carbonate or acetate are given to reduce phosphate and PTH levels
Vit/Min supplements usually prescribed:
Folic acid=1 mg/day; B6=10 mg/day; DRI for other B-complexes; C=60-100 mg/day; supplements w/active form vit. D given orally or IV; intermittent high doses calcitriol orally or by IV to suppress elevated PTH; Fe supplements usually necessary for pts. receiving EPO (periodic IV Fe may be needed if oral intake cannot maintain serum transferrin saturation >20-30% or serum ferritin levels >200-300 ng/mL)

Peritoneal Dialysis

1.2-1.5 g/kg/day protein for CAPD/CCPD pts. During periods of peritonitis, greater needs of protein d/t greater losses across inflamed peritoneum.
25-30 kcals/kg needed. 25-50 kcals/kg may be needed for repletion.
Fat is same as for hemodialysis
Na+ needs are about 2-4 g/day and fluid needs are about 2 L/day. Higher b/c dialysis done daily and therefore have more liberal intakes.
K+ usually 3-4 g/day and often not restricted, some even need more.
Patients may need some P restriction, but considering their increased protein needs, P intake cannot be <15 mg P/kg/day
Vitamin/Mineral supplements same as for hemodialysis. Pts. may not receive calcitriol.

Renal Transplant

1.3-2.0 g protein/kg/day is recommended d/t increased needs from surgery and steroid therapy. 1.2-1.5 g/kg/day as protein more reasonable and adjusted individually. 1.0 g/kg if chronic and stable.
30-35 kcals/kg dry or usual BW sufficient. Corticosteroid therapy leads to increased appetite, but weight maintenance is essential long-term. Monitor closely. Kcal restriction may be warranted for overweight pt's.
Hyperglycemia may occur from drug therapy. Those who develop glucose intolerance or DM need a calorie controlled diet to prevent excessive wt. Gain and control blood sugars. Avoid high glycemic foods (candy, juices, and honey). Increase exercise.
Limit fat to 30% of kcals and <300 mg/day cholesterol. Emphasize mono-polyunsaturated fats for long-term d/t increase risk of CHD b/c of immunosuppressive and hypertensive therapies that induce hyperlipidemia. Increasing omega-3 ffa's may benefit pt's on cyclosporine therapy. Good for general well-being also.
Na+ is ~2-4 g/day post-op and 3-4 g/day chronic and stable. Na+ and fluid retention may occur from corticosteroid therapy (restrict Na+ intake in this case). Fluid not restricted unless fluid overload from kidney rejection, edema, or ATN.
K+ not restricted unless hyperkalemia develops from cyclosporin therapy. Rejection of kidney or ATN may require K+ restriction also.
P restriction not needed usually if kidney functioning properly. HypoPatemia may occur in acute post-transplant period and high phosphate diet or P supplement may be needed. P BINDERS most often NOT NEEDED!!
1-1.5 g Ca++/day through food or supplement needed. Ca++ absorption may be disrupted d/t corticosteroid therapy (chronic post-transplant period)
Renal vit preparations still needed often temporarily in post-transplant pt. especially if dietary restrictions from ATN or rejection. Fe therapy may be continued if anemia present and EPO admin continued.

Things I may have missed

Avoid salt substitutes w/pt's who have K+ restriction
Pt's with urinary tract infection may benefit from cranberry juice
Dialysis pt's with:
High serum Na+ will need to reduce Na+ intake and make sure they are gaining 4% of BW b/t dialysis and not dehydrated. If Na+ low then pt. may need to reduce fluid intake.

High serum K+ levels will need to avoid foods w/250 mg K+/serving and limit total intake to 2 g K+/day. If low K+, adding one 250 mg K+ food per day may help (check frequently).
Chloride changes is directly related to amount of Na+ intake. No dietary changes.
If total CO2 is low, pt. may need more dialysis.
If creatinine changes, no dietary changes noted. Dialysis controls level.
Dialysis pt's need a minimum of 4 servings breads/starches and 2-3 servings fruit for energy. Avoid concentrated sweets (especially w/diabetics) unless hypoglycemic.
High Ca++ levels can be controlled by eating fewer dairy prods. If low, increasing Ca++ in the diet (if P is normal) by adding more dairy prods. Ca++ supplement or active Vit. D may be needed.
Limit milk and milk products to 1 serving/day if P is high. Take P binders as prescribed.
If P levels are low, add 1 serving milk product or other high P food/day and recheck levels
High BUN may need to limit intake of meats and dairy prods. To about three servings/day. If low, it may be low if not eating enough and losing weight. May increase w/muscle loss.
Increased uric acids-NO DIETARY CHANGE! Purines are found in most foods. If gout occurs see physician for drugs.
Keep Ca++ and P w/in NL levels if altered Alk Phos.
No dietary changes w/ change in LDH or SGOT
May need to limit cholesterol intake if high cholesterol. If low, increase foods rich in cholesterol.
If total protein is low, increase intake of high protein foods.
See doctor if Hct dropping.
If serum ferritin low, may need iron supplement. Do not take at same time w/P binders.
SEE YOUR DIETITIAN!!
Monitor RBP b/c elevated in pt's w/renal failure
Monitor trace minerals
Monitor blood glucose levels frequently w/pt's on TPN. Some pt's may benefit from a snack rich in complex CHO's towards end of treatment.
TPN solutions for dialysis pt's according to access routes:
NL TPN solution for hemodialysis pt's is 70% dextrose (350 g glucose), 15% a.acids (25 g pro), 20% lipid emulsion (50 g fat).
NL TPN solution for TPN by subclavian veins is 70% dextrose (700 g glucose), 15% a.acids (50 g pro), 20% lipid emulsion (100 g fat).
NL TPN solution for peripheral vein is 10% dextrose (50 g glucose), 10% a.acids (40-50 g pro), 10% lipid emulsion (50 g fat).