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PCCN CCRN

The Renal System

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Kidney Processes

- Filtration
- Diffusion
- Osmosis
- Secretion
- Excretion
- Reabsorption

Kidney Functions

- Urine Formation
- Excretion of Metabolic Wastes
  - Creatinine
  - Urea
- Fluid & Electrolyte Regulation
- Blood Pressure Control
- Red Blood Cell Synthesis
- Maintain Acid-Base Balance

Fluid Imbalances

- Hypervolemia
- Hypovolemia
- Dehydration third-spacing

Dehydration Third Spacing-Etiology

- Increased capillary permeability
- Lymphatic blockage
- Lowered plasma proteins

Phase 1: Loss

- Increased capillary permeability
- Shift of protein and fluid to the interstitial spaces
- Hypovolemia with pallor, cold extremities, apprehension, ↑HR, low BP, ↑HCT/HGB
- Management: crystalloids, monitor hemodynamic parameters and intake and output

Phase 2: Reabsorption

- Capillary repair with return of normal permeability
- Shift of fluid to intravascular spaces
- Hypervolemia with increased urine output, crackles, SOB
- Management: daily weights, monitor parameters and intake and output

Electrolyte Imbalance

- Fluid Loss
- Renal Failure
- Diuretics
- CHF
- Excess Water intake
- Acid-Base Imbalance
- Muscle cell damage
- Parathyroid and Adrenal disorders
- Excessive intake

Sodium Function

- Maintain osmotic pressure and serum osmolality
- Regulate fluid volume
- Maintain acid base balance
- Control muscle contraction

- Normal serum values: 136-145 mEq/L

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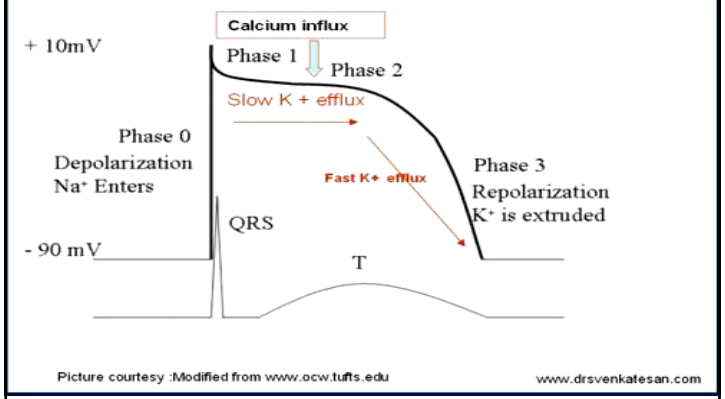
The hidden Ionic currents that generate the ECG



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Hyponatremia: Etiology

- Excess water intake: SIADH, Excess free water intake, cirrhosis, ARF with oliguria, injury with fluid resuscitation, sepsis
- Excess sodium loss: GI losses, Diaphoresis, diuresis, adrenal insufficiency, burns, hemorrhage

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Clinical Presentation- Water Excess

- Headache, weakness, convulsions
- Edema
- Weight gain
- High Blood pressure
- Dilute urine
- Nausea/vomiting

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Clinical Presentation: Dehydration

- Headache, confusion, convulsions, weakness
- Tachycardia, hypotension
- Dry mucous membranes
- Weight loss
- Concentrated urine
- Anorexia, nausea, vomiting

Treatment

- Dehydration: give high Na+ diet with adequate fluid intake and/or saline solutions
- Water excess: restrict fluids, diuretics

Hypernatremia: Etiology

- Excess water loss: diabetes insipidus, osmotic diuresis, inadequate fluid intake
- Excessive sodium intake: excessive intake of LR or NS, sodium bicarbonate
- Increased ECF volume with Na+ and water retention: CRF, HF, liver disease

Clinical Presentation: Dehydration

- Tremors, restlessness, irritability
- Low grade fever
- Dry mucous membranes
- Tachycardia, hypotension
- Thirst
- Oliguria

Clinical Presentation: Water Excess

- Edema
- Weight gain
- Hypertension
- Dyspnea, crackles
- Pulmonary edema

Treatment

- Dehydration: give free water
- Water excess: administer diuretics

Potassium: Function

- Maintain osmotic pressure of ICF
- Regulate neuromuscular excitability
- Assists to regulate acid-base balance

- Normal serum values: 3.5-5 mEq/L

Hypokalemia: Etiology

- Transcellular shift in alkalosis
- Increased aldosterone, large doses of cortisol, insulin
- Inadequate K+ intake
- Excessive loss of K+: GI loss, diuretics, Mg++ deficiency, laxative abuse

Clinical Presentation

- Shallow respirations
- Drowsiness to coma
- Muscle weakness progressing to paralysis
- Dilute urine, polyuria
- Abdominal distention, N/V, ileus
- Dysrhythmias; ventricular ectopy, ST depression, inverted T waves, U waves
- Enhanced digitalis effect

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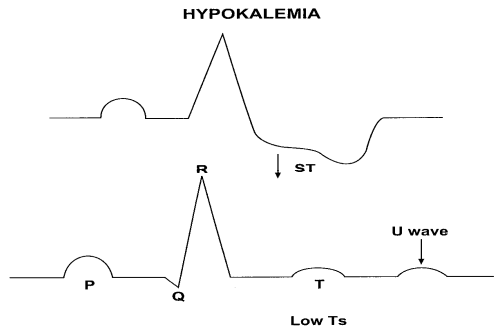
ECG Changes with Hypokalemia



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Treatment

- IV: KCL slowly IV (10-20mEq/hr) or PO repletion
- Treat underlying cause
- Cardiac monitoring

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Hyperkalemia: Etiololgy

- Increased K+ load: transcellular shift in acidosis, hyperglycemia; cellular damage or death (rhabdomyolysis, burns, trauma)
- Increased K+ intake (excessive IV KCL)
- Inability to excrete K+: Hypoaldosteronism, Renal failure (acute or chronic)
- Drugs: ACEI, NSAIDS, digoxin overdose, succinylcholine

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Clinical Presentation

- Dysrhythmias: bradycardia, asystole
- Peaked T wave, widened QRS, prolonged QTI, flattened P wave, ST depression
- Lethargy, confusion
- Weakness, numbness of extremities
- Oliguria
- Shallow respirations or deep rapid respirations (acidosis)
- N/V, diarrhea

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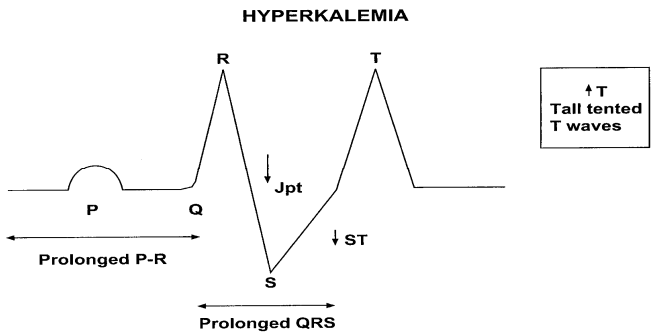
ECG Changes with Hyperkalemia



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Treatment

- Determine and treat cause
- Cardiac monitoring
- Stop K+ infusion
- Glucose, insulin
- NaHCO3
- CaCl
- Kayexalate/Sorbitol
- Dialysis

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Calcium: Function

- Bone formation and metabolism
- Stabilizes cell membrane
- Maintains normal transmission of nerve impulses and muscle contraction
- Coagulation
- Normal serum values: 9-11 mg/dL

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Hypocalcemia: Etiology

- Decreased absorption: small bowel resection, crohn's disease, biliary obstruction, alcoholism
- Increased excretion: diuretics, antacid overuse or laxatives
- CRF: increased phosphate
- Decrease in ionized Ca++: blood transfusions, alkalosis, hypoparathyroidism, pancreatitis, hypomagnesemia

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Clinical Presentation

- Muscle tremors, hyperactive reflexes; tingling around mouth, hands, feet
- +Trousseau sign, +Chvostek sign
- Bronchospasm
- Prolonged ST segment, QT interval, torsades de pointes
- Impaired clotting: bruising, bleeding
- Labored shallow breathing
- Abdominal cramping, N/V, diarrhea

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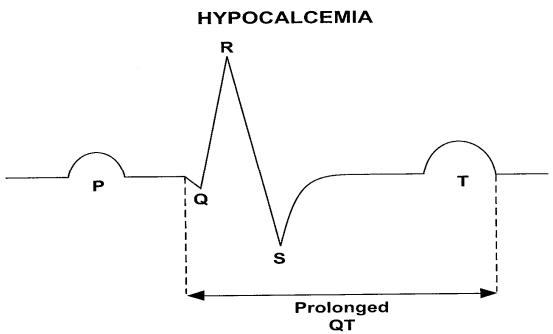
ECG Changes with Hypocalcemia



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Treatment

- Identify cause and treat
- Cardiac monitoring
- IV 10% calcium gluconate or calcium chloride

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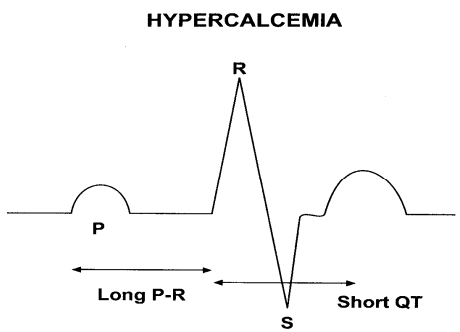
Hypercalcemia: Etiology

- Increased mobilization from bones: immobility, malignancy, hyperparathyroidism, thyrotoxicosis, hypophosphatemia
- Increased intake: dietary, excessive Vitamin D, excessive antacid use
- Altered reabsorption: chronic thiazide diuretics, increased parathyroid hormone

Clinical Presentation

- Diminished reflexes, weakness, drowsiness
- Decreased LOC
- Anorexia, N/V, constipation
- Flank pain: renal calculi
- Dysrhythmias: Decreased ST segment, short QT interval, heart blocks
- Enhanced digitalis effect
- Pathologic fractures

ECG Changes with Hypercalcemia



Treatment

- Determine and treat cause
- Cardiac monitoring
- NS infusions/diuretics to increase secretion
- Corticosteroids to block absorption
- Pilcamycin
- Phosphorus

Magnesium: Function

- Affects protein and CHO metabolism
- Affects neuromuscular transmission and contractility
- Influences transport of Na+ and K+ across cell membrane
- Influences parathyroid hormone release

- Normal serum values: 1.3-2.1 mEq/L

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Hypomagnesemia: Etiology

- Impaired absorption: malabsorption syndromes, alcoholism, bowel resection, acute pancreatitis
- Increased excretion: diuretics, diuresis, diarrhea, increased aldosterone
- Hypothermia, sepsis

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Clinical Presentation

- Tremors, seizures, confusion
- Muscle weakness, hyperreflexia
- +Chvostek's sign
- +Trousseau sign
- Decreased LOC, psychosis
- Tachycardia, hypotension
- Dysrhythmias: ventricular ectopy, depressed T waves, ST depression, prolonged QT interval (PVC's, vfib, SVT)
- Risk of digitalis toxicity

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Treatment

- Mg+ 1-2 grams over 1 hour (1 gm/50mL)
- Mg+ 3-4 grams over 2 hours (2-3 gms/100mL)
- Infuse 150 mg/min
- Monitor for hypotension, respiratory/CNS depression

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Hypermagnesemia: Etiology

- Excessive IV or PO intake: Mg+ antacids or laxatives containing Mg+, treatment of eclampsia
- Inadequate renal excretion: renal failure
- Untreated DKA
- Hypothyroidism, hyperparathyroidism
- Rhabdomyolysis

Clinical Presentation

- Drowsiness, lethargy, weakness
- N/V
- Depressed respirations
- Hypotension, ↓HR, vasodilation
- Prolonged PR, QRS, QT interval
- Peaked T waves
- Heart Block
- Decreased deep tendon reflexes

Treatment

- Determine and treat cause
- Calcium (antagonist to Mg+)
- Fluids and diuretics
- Dialysis

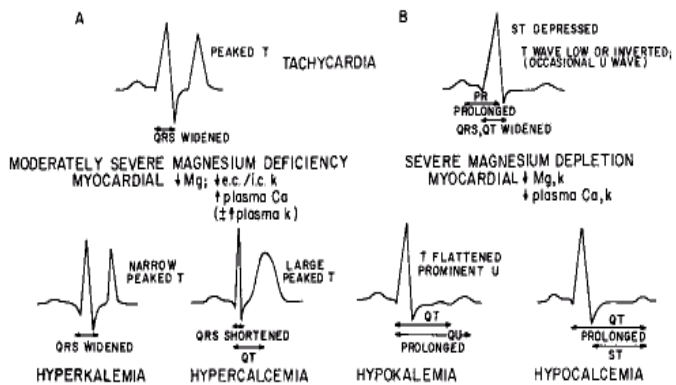

FIGURE 9-1. Schematic representations of electrocardiograms of moderate and severe hypomagnesemia, compared to those of hyper- and hypokalemia and -calcemia. (From Seelig, 1969.)

Hypokalemia can be caused by which of the following?

a. Crush injuries and nasogastric suction

b. Blood transfusions and hemolysis

c. Diuretics and alkalosis

d. Diarrhea and acidosis

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Acute Renal Failure

- Defined as a measurable increase in serum creatinine concentration by usually 50%.
- Sudden deterioration in renal function resulting in decreased GFR
- Types: Pre-renal, Intrarenal, Post-renal

Etiology

- Anything that diminishes renal blood flow!

Volume

– Decreased intravascular volume: internal fluid shifts (burns, peritonitis, ileus, third spacing), external fluid shifts (hemorrhage, vomiting, diarrhea, diuresis)

Cardiac Pump

– Cardiovascular failure: decreased CO (cardiogenic shock, dysrhythmias, MI, HF, pulmonary embolism)

Distributive

– Vasodilation: sepsis, vasodilating drugs

Laboratory

- Urinary Na+ < 10 mEq/L
- FEna <1%
- Specific gravity > 1.015
- Elevated BUN
- Slightly elevated creatinine
- Urine osmolality > 500 mOsm (concentrated)
- Minimal or no proteinuria
- Normal urinary sediment

Treatment

- Fluids
- Diuretics (lasix, mannitol)
- Volume expanders
- Improve CO (enhance contractility and optimize filling pressures)

Intrarenal

- Damage to glomeruli, vessels or tubules
- Types: cortical and medullary (acute tubular necrosis)

Etiology

- Cortical: vascular, infectious or immunological processes cause renal capillary swelling and cellular proliferation → obstruction of glomerulus by edema or cellular debris → ↓ GFR → ↓ urine output → oliguria
- Medullary (ATN): most common type seen in ICU's

Medullary

- Nephrotoxic injury (epithelial layer)
  - **Antibiotics (cephalosporins, penicillins, tetracyclines)**
  - **Pesticides and fungicides**
  - **Contrast dyes**
- Ischemic injury (tubular basement membrane)

  MAP below 60 mm Hg for over 40 min.
  - **Massive hemorrhage**
  - **Transfusion reaction**
  - **Cardiogenic shock**
  - **Major trauma or crush injuries**

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Laboratory Values - Cortical

- Urinary Na+ < 10 mEq/L
- FEna >2%
- Specific gravity varies
- Moderate-to-heavy proteinuria
- Serum BUN and creatinine elevated
- Hematuria
- Urinary sediment with erythrocyte casts and leukocytes

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Laboratory Values - Medullary

- Urinary Na+ > 20 mEq/L
- Specific gravity < 1.010
- Minimal-to-moderate proteinuria
- Serum BUN and creatinine elevated
- Urinary sediment with renal tubule epithelial cells, tubular casts, and a rare erythrocyte

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Pre-Renal

- Diminished perfusion to kidney without renal tubular damage

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Phases

- 1. Onset or initial phase
- 2. Oliguric phase or Nonoliguric phase
- 3. Diuretic phase
- 4. Recovery

Post-Renal

- Obstruction of urinary collecting system
- Partial or complete obstruction occurs anywhere from kidney to urinary meatus (urethral obstruction, prostatic hypertrophy, blood clots, tumor, renal stones)
- Obstruction causes ↑ pressure in tubules, ↓ GFR, and oliguria

Clinical Presentation

- Oliguria or anuria
- Hypervolemia (CHF, cerebral edema, crackles, confusion)
- Laboratory values: elevated BUN and creatinine specific gravity varies, urinary sediment (RBCs, WBCs, uric acid crystals, hyaline casts)

Management

- Remove obstruction followed by close monitoring for post-obstructive diuresis
- Watch for hypovolemia in first 24-48 hours
- Monitor electrolyte and acid-base balance
- If no diuresis, suspect renal damage

Clinical Presentation of Uremia

- Confusion, lethargy, twitching, and weakness related to metabolic acidosis
- Nausea, vomiting, anorexia, coffee ground emesis, melena, abdominal distension, diarrhea or constipation
- Deep rapid respirations secondary to metabolic acidosis, crackles

Clinical Presentation

- Dysrhythmias (secondary to electrolyte imbalances)
- Pulmonary edema, pericarditis (↑HR, ↓BP,↑RR)
- Hypertension
- Oliguria or nonoliguria
- Electrolyte imbalances
- Altered excretion of medications
- Dry skin, uremic frost, pruritis, pallor, bruising, edema

Clinical Presentation

- Increased risk of infection
- Decreased erythropoietin
- Thrombocytopenia
- Hypermetabolic

In a patient with ATN, which of the following should be expected?

a. Hypercalcemia, hypertension, and acidosis

b. Hypokalemia, anemia, and hypertension

c. Hyperkalemia, acidosis, and azotemia

d. Hypocalcemia, anemia, and alkalosis

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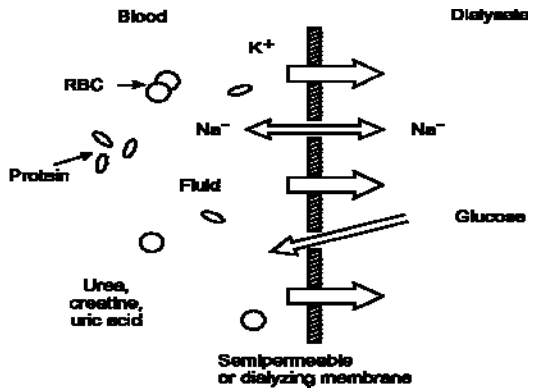
Dialysis

- Diffusion of dissolved particles from one fluid compartment to another across a semi-permeable membrane
- Principles: osmosis, diffusion, filtration

Osmosis & Diffusion In Dialysis



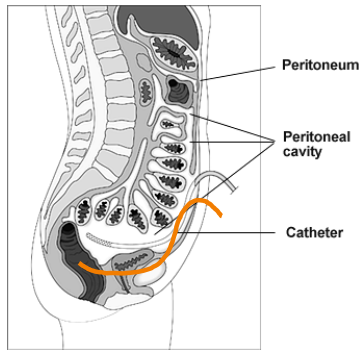
Types of Dialysis

- Peritoneal
- Hemodialysis
- Continuous Renal Replacement Therapies (CRRT)

Peritoneal Dialysis

- Peritoneum is semi-permeable membrane
- Blood is one fluid compartment, dialysate the other
- Performed manually or by machine

Peritoneal Dialysis



Indications

- Fluid overload
- Electrolyte or acid-base imbalance
- Acute or chronic renal failure
- Unavailability of vascular access for hemodialysis
- Inability to anticoagulate

Advantages

- Less complicated equipment
- Less need for highly skilled personnel
- Availability of supplies and equipment
- Less stressful for elderly and pediatric patients
- Anticoagulation not required

Disadvantages

- Requires more time to adequately remove metabolic wastes and restore electrolyte and fluid balance
- Repeated treatments may lead to peritonitis

Contraindications

- Abdominal adhesions
- Recent abdominal surgery
- Acute peritonitis
- Coagulopathy
- Pregnancy

Procedure

- Weigh patient before and after procedure
- Select dialysate solution (more glucose pulls off more fluid – 1.5%, 2.5%, 4.25%)
- Warm solution
- Add prescribed medications (heparin, KCL, antibiotics, lidocaine)
- Ensure bladder empty

Procedure

- Catheter inserted by physician (midline and below umbilicus)
- Start infusion (1-3 L over 10-20 min)
- Dwell time 20-45 min
- Drain at end of dwell time

Effluent Color

- Normal: clear, pale yellow
- Cloudy: infection
- Brownish: bowel perforation
- Amber: bladder perforation
- Blood-tinged: normal in first 1-4 exchanges; if it continues, suspect abdominal bleeding

Management

- Monitor intake and output (fluid out should exceed fluid in)
- Monitor VS for hypo/hypervolemia
- Monitor for technical difficulties
- Maintain closed system
- Obtain periodic culture of effluent

Complications

- Peritonitis/infection (fever, abdominal tenderness, elevated WBC's)
- Respiratory distress
- Perforation of bowel or bladder

Hemodialysis

- Blood is one fluid compartment and dialysate the other with an artificial semi-permeable membrane
- Blood pump, dialyzer, dialysate, vascular access
- Anticoagulation

Indications

- Acute or chronic renal failure
- Rapid removal of fluid, toxins, poisons or drugs
- Removal of waste products
- Electrolyte imbalance
- Contraindication to peritoneal dialysis
- Removal of excess water through ultrafiltration

Contraindications

- Hypotension
- Hypovolemia
- Hemodynamic instability
- Coagulapathy
- Do not administer dialyzable drugs or drugs that may cause hypotension immediately before hemodialysis

Access

- Shunts: AV access, surgical procedure, can be used immediately, long term access
- Fistula: anastomosis of an artery and vein, 4-6 weeks until functional, long term access
- Short term: femoral venous catheter; immediate venous access; monitor distal pulses; bedrest
- Short term: subclavian or jugular venous catheter; immediate venous access; monitor for pneumothorax

Complications

- Hypotension
- Air embolism
- Dysrhythmias from fluid shifts and electrolyte changes
- Hemorrhage
- Infection
- Disequilibrium syndrome (too rapid removal of waste products)

Continuous Renal Replacement Therapy (CRRT)

- Continuous ultrafiltration (removal of fluid) and clearance of uremic toxins in a slower, more controlled manner

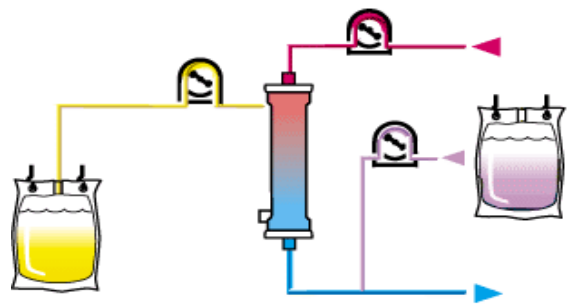
Indications

- Hemodynamic instability (ARF, pulmonary edema, HF, AMI, sepsis, MODS)
- Oliguria in patients who require large amounts of parenteral fluid (TPN, vasopressors, IV antibiotics) not responsive to diuretics
- Inability to tolerate hemodialysis

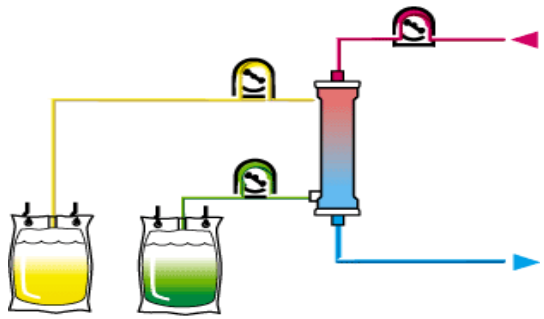
Types

- Slow continuous ultrafiltration (SCUF)
- Continuous arteriovenous hemofiltration (CAVH)
- Continuous arteriovenous hemodialysis (CAVHD)
- Continuous venovenous hemofiltration (CVVH)
- Continuous venovenous hemodialysis (CVVHD)
- Continuous venovenous hemodifiltration (CVVHDF)

Continuous Venovenous Hemofiltration (CVVH)



Continuous Venovenous Hemodialysis (CVVHD)



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Selection

- Based on fluid balance, metabolic state, electrolyte balance, vascular access, and blood pressure

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Advantages

- Fluid removal without osmolar changes
- Decreased risk of hemodynamic instability
- No reduction in platelet and WBC count
- Can be managed by ICU RN at bedside

Important to note that this is not an emergency therapy for overdose, fluid or waste product removal.

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Complications

- Hypotension
- Hypo/hypervolemia
- Electrolyte imbalance
- Access complications: bleeding, clotting, infection
- Hemorrhage from anticoagulation, disruption of filter or tubing
- Hypothermia

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Chronic Renal Failure

- Gradual (several months or several years) loss of renal function
- Kidneys have the ability to adapt to a decreasing number of functioning nephrons
- Able to function with less than 25% of original nephrons

Etiology

- Diabetes mellitus
- Glomerulonephritis
- Polycystic disease

Diabetes Mellitus

- Progressive
- Small vessels of kidney thicken
- Results in decreased blood flow, nephron destruction, and proteinuria
- ACE inhibitors (Captopril) slow the progression

Glomerulonephritis

- Chronic inflammation of glomeruli from streptococcal infection
- Once started, little can be done to stop the process
- Varying degrees of proteinuria, hematuria, hypertension, and urinary sediment
- Process can be slowed with BP control and dietary protein restriction

Polycystic Disease

- Genetic disorder characterized by multiple cysts that enlarge and compress the kidney
- Renal function compromised when person 40-50 years old
- Hypertension seen prior to renal failure as compression by the cysts promotes renin secretion
- Hematuria (from ruptured, bleeding cysts), flank pain, abdominal pain
- Nephrectomy may be necessary

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Stages of CRF

- Diminished renal reserve
- Renal insufficiency
- End-stage renal disease
- Uremic syndrome

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Diminished Renal Reserve

- GFR 40-50% of normal (50% nephron loss)
- Serum BUN and creatinine normal or high normal
- Creatinine doubles (1.4-2.4)
- Patient asymptomatic

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Renal Insufficiency

- GFR 20-40% of normal (75% nephron loss)
- Serum BUN and creatinine elevated
- Creatinine quadruples (5.6-9.6)
- Mild anemia, mild azotemia
- ↑phosphorus, ↑potassium, ↓bicarbonate
- SG low (impaired ability to concentrate urine)
- Progression influenced by severity of hypertension, dietary protein intake, infection, cardiac failure, and nephrotoxic drugs

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End-Stage Renal Disease

- GFR < 10% of normal (90% nephron loss)
- Serum BUN and creatinine very elevated
- Anemia, azotemia, metabolic acidosis, fluid and electrolyte abnormalities, oliguria
- Renal replacement therapies needed to maintain life

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Uremic Syndrome – Clinical Presentation

- Complete nephron loss
- Anemia, thrombocytopenia, immunosupression
- Hypervolemia, hypertension, ↑ rate of atherosclerosis, pericarditis, ↑HR, dysrhythmias
- Anorexia, nausea, vomiting, ulcers, diarrhea, constipation, bleeding
- Lethargy, fatigue, “restless leg syndrome” (numbness, burning, cramping in feet/legs at night), peripheral neuropathy

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Clinical Presentation

- Oliguria, proteinuria, casts and cells in urine
- Pruritis, uremic frost, pallor
- Metabolic acidosis, ↑↓ Na+, ↑ K+, ↓ Ca+, ↑Mg+, hyper/hypovolemia
- Changes in menstral cycle, decreased libido, impotence
- Renal osteodystrophy from ↑ phosphate/ ↓ calcium, and metabolic acidosis (joint pain, gait changes, muscle weakness)

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Management

- Fluid control (sodium and water restriction, dietary teaching)
- Electrolyte control (decrease K+ intake, eliminate dairy products, restrict protein, dialysis)
- Dietary control (calories from CHO and fat)
- Epoetin alfa (EPO)
- Quality of life

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Renal Trauma

- Nonpenetrating injuries (80% of all renal injuries)
- Penetrating injuries (20% of all renal injuries)

Classification

- Contusion
- Lacerations
- Fractures
- Vascular

Renal contusion

Deep laceration

Segmental infarction

Global infarction

Assessment

- Pain in flank, upper quadrant of abdomen, orcostovetebral angle
- Hematoma over or near kidneys
- Hematuria
- Entrance wound
- Retroperitoneal bleeding
- Blood or ecchymosis over genitalia, and perineum

Diagnostic Studies

- ↑BUN, ↑creatinine
- ↓Hgb/Hct
- Electrolytes levels vary
- Urine volume varies
- Urinalysis may be normal
- Hemturia

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Diagnostic Studies

- KUB
- IVP
- Ultrasound
- Renal scan
- Renal angiography
- Surgical exploration

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Complications

- Hemorrhage (rebleed or delayed bleeding)
- Extravasation of urine
- Abscess
- Ileus
- Sepsis, shock
- Late complications: hypertension, hydronephrosis, chronic pyelonephritis

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Management

- Minor injury: bedrest x 10 days without strenuous activity x 3 weeks
- Major injury: monitor for shock!
- Extravasation of urine

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Surgical Intervention

- Indication: shattered kidney, vascular injuries, expanding or pulsatile hematoma, extravasation of urine, continually decreasing Hct
- Post-op: analgesics, monitor intake and output, serum studies, and vital signs