A Four Dimensional Approach to Acute Renal Failure
The track of understanding renal physiology takes us through a convoluted journey
WHAT'S IT ALL ABOUT?

EAT, SURVIVE, REPRODUCE.

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EAT, SURVIVE, REPRODUCE.
**Electrolytes** are charged particles (ions) that are dissolved in body fluids.

**Electrolytes (Dissolved Ions)**

**Major Positive Ions (Cations)**
- $\text{Na}^+$ Sodium ion, Na$^+$
- $\text{K}^+$ Potassium ion, K$^+$
- $\text{Ca}^{2+}$ Calcium ion, Ca$^{2+}$
- $\text{Mg}^{2+}$ Magnesium ion, Mg$^{2+}$

**Major Negative Ions (Anions)**
- $\text{Cl}^-$ Chloride ion, Cl$^-$
- $\text{HCO}_3^-$ Bicarbonate ion, HCO$_3^-$
- $\text{HPO}_4^{2-}$ Phosphate ions, HPO$_4^{2-}$ & H$_2$PO$_4^-$
- $\text{SO}_4^{2-}$ Sulfate ion, SO$_4^{2-}$
- Organic acids
- Proteins
Intracellular Milieu

- High Potassium
- High Protein
- High Magnesium
- High Phosphate

Very low sodium and chloride
Very low bicarbonate
PROFOUNDLY low calcium
The Inside of the Cell is a Protein rich, high potassium, high phosphate, and high magnesium environment

...this is where the processes of creating energy and life take place!!
The Outside of the Cell is the Salt of the Sea from which Life Sprang Forth

...bathing the cells in the liquid from which they were born
Intracellular Fluid (ICF) 20 liters
- 40% Body Weight (24 Liters)

Interstitial Fluid (IF) 10 liters
- 16% Body Weight (9.6 Liters)

Plasma 5 liters
- 4% Body Weight (2.4 Liters)

Extracellular Fluid
## Body Fluid Compartments

<table>
<thead>
<tr>
<th></th>
<th>Body Weight (%)</th>
<th>Total Body Water (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total</strong></td>
<td>60</td>
<td>100</td>
</tr>
<tr>
<td><strong>Intracellular</strong></td>
<td>40</td>
<td>67</td>
</tr>
<tr>
<td><strong>Extracellular</strong></td>
<td>20</td>
<td>33</td>
</tr>
<tr>
<td><strong>Intravascular</strong></td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td><strong>Interstitial</strong></td>
<td>15</td>
<td>25</td>
</tr>
<tr>
<td>Age</td>
<td>Total Body Water As % Body Weight</td>
<td>Extracellular Fluid As % Body Weight</td>
</tr>
<tr>
<td>-----------------</td>
<td>----------------------------------</td>
<td>-------------------------------------</td>
</tr>
<tr>
<td>Premature</td>
<td>75 - 80</td>
<td>50</td>
</tr>
<tr>
<td>Newborn</td>
<td>70 – 75</td>
<td>50</td>
</tr>
<tr>
<td>1 Year Old</td>
<td>65</td>
<td>25</td>
</tr>
<tr>
<td>Adolescent Male</td>
<td>60</td>
<td>20</td>
</tr>
<tr>
<td>Adolescent Female</td>
<td>55</td>
<td>18</td>
</tr>
</tbody>
</table>
### Electrolyte Concentration in Fluid Compartments (meq/L)

<table>
<thead>
<tr>
<th>Cations</th>
<th>Plasma</th>
<th>Interstitial Fluid</th>
<th>Intracellular Fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺</td>
<td>140</td>
<td>146</td>
<td>12</td>
</tr>
<tr>
<td>K⁺</td>
<td>4</td>
<td>4</td>
<td>150</td>
</tr>
<tr>
<td>Ca²⁺</td>
<td>5</td>
<td>3</td>
<td>10⁻⁷</td>
</tr>
</tbody>
</table>
## Electrolyte Concentration in Fluid Compartments (meg/L)

<table>
<thead>
<tr>
<th></th>
<th>Plasma</th>
<th>Interstitial Fluid</th>
<th>Intracellular Fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anions</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cl⁻</td>
<td>103</td>
<td>114</td>
<td>3</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>24</td>
<td>27</td>
<td>10</td>
</tr>
<tr>
<td>SO₄²⁻</td>
<td>1</td>
<td>1</td>
<td>---</td>
</tr>
<tr>
<td>HPO₄³⁻</td>
<td>2</td>
<td>2</td>
<td>116</td>
</tr>
<tr>
<td>Protein</td>
<td>16</td>
<td>5</td>
<td>40</td>
</tr>
<tr>
<td>Organic anions</td>
<td>5</td>
<td>5</td>
<td>---</td>
</tr>
</tbody>
</table>
Nurse Fanny say:
"You can kiss that urinal good-bye!"
Water is constantly being lost

- Urine
- Feces
- Sweat
- Spit (south Georgia)
Exercise, fever, vomiting, diarrhea. NG suction, other tubes...

All remove volume from the body, initially from the circulating vascular volume, then from the interstitium and cells.
## Water Losses
### in a 60 – 80 Kg Man

<table>
<thead>
<tr>
<th></th>
<th>Average Daily Volume (mL)</th>
<th>Minimal Daily Volume (mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sensible Losses</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urinary</td>
<td>800 - 1500</td>
<td>300</td>
</tr>
<tr>
<td>Intestinal</td>
<td>0 - 250</td>
<td>0</td>
</tr>
<tr>
<td>Sweat</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Insensible Losses</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lungs and Skin</td>
<td>600 – 900</td>
<td>600 - 900</td>
</tr>
</tbody>
</table>
The nephron leaks out 180 liters/day

...that is 60 times the normal adult's plasma volume!!!
We literally pour out our entire PLASMA VOLUME every 30 minutes

...that’s a powerful organ!
Yet, the “beans” reabsorb some 99% of this volume

...that is some refining power
With a GFR of 120 ml/min (180 L/day) 25,000 mEq of sodium (about half a kilogram) are filtered out per day, almost all of which are reabsorbed.
A “Trip through the Tubules”
Tubular fluid is iso-osmolar in the proximal tubule
The Renal Medulla is EXCEEDINGLY concentrated and highly osmotically concentrated...

...2 – 4 times the osmolarity of the filtrate.
Water, then, is reabsorbed in the descending loop of Henle.
In the ascending loop, Na / K / Cl are reabsorbed...

...this portion is impermeable to water, even with ↑↑ ADH
Tubular fluid becomes more dilute as it flows up the ascending loop.

...osmolarity of 100 mOsm/L
In the Distal Tubules and Collecting Ducts, fluid may become even more dilute due to additional reabsorption of NaCl. 

...impermeable to water in ABSENCE of ADH (osmolarity ↓ to 50 mOsm/L)
Urine Sodium is the “signal” of what the body is trying to do ...unless the kidneys are failing
Look at the BODY...

...then look at the SIGNAL!
Look at Hydration and Hemodynamic Status

...and then look at the fluid being eliminated
How effective is the SIGNAL?

*We can excrete as little as 1 meq Na\(^+\) per day*

*...or as much as 5000 meq Na\(^+\)*
How effective is the SIGNAL?

We can dilute urine to as little as 50 mOsm/L of solute in 300 cc urine

...or concentrate as much as 1400 mOsm/L
It’s what 2 million nephrons with multiple hormonal signals will do for you

…and, it’s what an organ that processes 36 times your blood volume per day can do
The renal excretion of water is independent of solute excretion

...the primary effector or water excretion is ADH (vasopressin)
When Osmolarity is greater than normal, the posterior pituitary secretes ADH.

...increases permeability of the distal tubules and collecting ducts to water.
When Osmolarity is greater than normal, the posterior pituitary secretes ADH

- ↑ Water absorption
- ↓ Urine volume
- Does not markedly alter rate of renal excretion of solute
When Osmolarity is less than normal, the posterior pituitary secretes less ADH...

...reduces the permeability of distal tubules and collecting ducts to $H_2O$
Kidneys can excrete up to 20 liters/day of dilute urine with Osmolarity as low as 50 mOsm/L (serum 285 - 295)
After drinking a liter of H\textsubscript{2}O, within 45 minutes urine volume ↑ by 600% … with urine concentration falling from 600 mOsm/L to 100 mOsm/L
The kidneys can concentrate the urine to 1400 mOsm/L

...desert animals can concentrate urine to 10,000 mOsm/L, surviving in the desert without drinking water, getting water only from food
Sea water has 2400 mOsm/L

…but the lowly Australian hopping mouse can excrete the salt load in a liter of sea water in 200 cc of urine and could thus drink sea water with impunity.
A normal human excretes 600 mOsm of solute per day

...if maximum concentrating ability is 1200 – 1400 mOsm/L, then: 600/1200 = 0.5 L/day minimal urine volume to excrete normal solute waste
# Electrolyte Requirements

<table>
<thead>
<tr>
<th>Electrolyte</th>
<th>Requirement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium</td>
<td>1.0 – 1.5 mEq/Kg (43.4 mEq/gram)</td>
</tr>
<tr>
<td>Potassium</td>
<td>0.5 – 0.75 mEq/Kg (~ ½ Sodium)</td>
</tr>
<tr>
<td>Chloride</td>
<td>1.0 – 1.5 mEq/KG</td>
</tr>
<tr>
<td>Solution</td>
<td>Glucose (g/L)</td>
</tr>
<tr>
<td>-------------------------</td>
<td>---------------</td>
</tr>
<tr>
<td>5% Dextrose (D5W)</td>
<td>50</td>
</tr>
<tr>
<td>10% Dextrose (D10W)</td>
<td>100</td>
</tr>
<tr>
<td>Normal Saline (NS)</td>
<td>0</td>
</tr>
<tr>
<td>D5NS</td>
<td>50</td>
</tr>
<tr>
<td>D5 ½ NS</td>
<td>50</td>
</tr>
<tr>
<td>0.2% NS</td>
<td>0</td>
</tr>
<tr>
<td>3% NaCl</td>
<td>0</td>
</tr>
<tr>
<td>Ringer’s Lactate (RL)</td>
<td>0</td>
</tr>
<tr>
<td>D5 RL</td>
<td>50</td>
</tr>
</tbody>
</table>
Urine Sodium is the “sign” of what the body is trying to do …unless the kidneys are failing…
Fractional excretion of sodium

The fraction of sodium actually excreted by the body relative to the amount filtered by the kidney.
Fractional excretion of sodium

\[
\text{Fe}_{\text{Na}} = \frac{U_{\text{Na}} / P_{\text{Na}}}{U_{\text{Cr}} / P_{\text{Cr}}}
\]
Fractional excretion of sodium

\[ \text{FE}_{\text{Na}} \text{ should be } 1\% - 3\%. \]

Anything higher than 3\% indicates impaired tubular function.

Diuretics \textbf{MAY} elevate this number.
...which brings us to...
Acute Renal Failure

A Proliferation of Physiological Failures Leading to Renal Malfunction
Acute Renal Failure:

- Definition
- Acute Renal Failure vs. Chronic Renal Failure
- Etiology
  - Pre-renal causes
  - Intrinsic Renal Diseases
- Acute Tubular Necrosis
- Presentation of Renal Failure
- Evaluation of the Patient
- Management
Acute Renal Failure:

Definition:

a. A sudden/severe decline in renal function resulting in accumulation of nitrogenous waste products

b. On a continuum with azotemia and renal insufficiency

c. May be accompanied by severe metabolic derangements – metabolic acidosis, volume overload, and hyperkalemia
Acute Renal Failure:

Acute vs. Chronic Renal Failure
a. Requires review of past history, records or labs
b. Important because aim in ARF is to reverse RF, aim in CRF is to treat complications
c. Consider that ARF may be superimposed on CRF
<table>
<thead>
<tr>
<th>Type of ARF</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prerenal Azotemia</td>
<td>• Hypovolemia</td>
</tr>
<tr>
<td></td>
<td>• Hypoalbuminemia</td>
</tr>
<tr>
<td></td>
<td>• Heart Failure</td>
</tr>
<tr>
<td></td>
<td>• Hypotension</td>
</tr>
<tr>
<td></td>
<td>• Renal artery/vein disease</td>
</tr>
<tr>
<td>Intrinsic Renal Disease</td>
<td>• Vasculitis</td>
</tr>
<tr>
<td>Vascular</td>
<td>• Scleroderma</td>
</tr>
<tr>
<td></td>
<td>• Malignant HTN</td>
</tr>
<tr>
<td>Glomerular</td>
<td>• Glomerulonephritis</td>
</tr>
<tr>
<td></td>
<td>• SLE</td>
</tr>
<tr>
<td></td>
<td>• Goodpasture’s</td>
</tr>
<tr>
<td>Interstitial</td>
<td>• Drugs</td>
</tr>
<tr>
<td></td>
<td>• Toxins</td>
</tr>
<tr>
<td></td>
<td>• Infections</td>
</tr>
<tr>
<td>Acute Tubular Necrosis</td>
<td>• Post Ischemia (post Prerenal)</td>
</tr>
<tr>
<td></td>
<td>• Myoglobinuria/hemoglobinuria</td>
</tr>
<tr>
<td></td>
<td>• Toxins (iodine contrast, aminoglycosides)</td>
</tr>
<tr>
<td>Post Renal Obstruction</td>
<td>• Extrarenal (tumor, neurogenic, urethral calculi or stricture)</td>
</tr>
<tr>
<td></td>
<td>• Intrarenal – bilateral or affecting single functioning kidney (calculi, tumor, papillary necrosis)</td>
</tr>
</tbody>
</table>
Acute Renal Failure:

Prerenal causes – Due to low blood flow to the kidney

a. Systemic Disease
b. Renal Artery/Vein Disease
Acute Renal Failure:

Systemic Disease

a. Hypovolemia
b. Hypotension
c. Third spacing of fluids
d. Congestive heart failure
e. Hypoalbuminemia – cirrhosis, nephrotic syndrome
Acute Renal Failure:

Renal Artery/Vein Disease
(some classify as *Intrinsic Renal Disease*)

a. Decreased blood flow
   (1) ACE inhibitors in the setting of bilateral renal artery stenosis
   (2) Prostaglandin Inhibitors – NSAID’s and ASA cause renal artery constriction

b. Artery obstruction (disease must be bilateral or affect the single functioning kidney)
   (1) Trauma
   (2) Aortic Aneurysm/Dissection
   (3) Tumor compression
   (4) Thromboembolic Disease
Acute Renal Failure:

Intrinsic Renal Disease – 5-10% of ARF in adults, 40-60% of ARF in pediatrics

a. Vascular
   (1) Malignant Hypertension
   (2) Scleroderma
   (3) TTP/Hemolytic Uremic Syndrome (HUS)
Acute Renal Failure:

Intrinsic Renal Disease – 5-10% of ARF in adults, 40-60% of ARF in pediatrics

b. Glomerular

(1) Systemic Vasculitis (polyarteritis, Wegener’s, HSP)
(2) Goodpasture’s Syndrome
(3) SLE
(4) Glomerulonephritis (Immune complex, post-strep, and rapidly progressive glomerular nephritis)
Acute Renal Failure:

**Intrinsic Renal Disease** – 5-10% of ARF in adults, 40-60% of ARF in pediatrics

c. **Interstitial**

(1) **Drugs** – probably immune related, not dose dependent, reoccurs with repeat exposure
   a. Penicillins
   b. Diuretics
   c. NSAIDS

(2) **Toxins**
   a. Heavy Metals
   b. Ethylene Glycol

(3) **Infections** – probably immune related
   a. Bacterial
   b. Rickettsia
Acute Renal Failure:

**Acute Tubular Necrosis** – most common cause of ARF in adults

1. Post – Ischemic
   Most commonly results from severe pre-renal azotemia

2. Nephrotoxins
   a. Antibiotics – aminoglycosides
   b. NSAIDS
   c. Iodine contrast
   d. Myoglobinuria
   e. Hemoglobinuria

3. Exclude pre-renal, post-renal, and intrinsic disease if ARF continues despite treatment
Presentation of Acute Renal Failure:

No particular clinical presentation is unique to Acute Renal Failure

1. Volume Status
   - Hypovolemic
   - Euvolemic
   - Volume overload
Presentation of Acute Renal Failure:

2. Azotemia
   a. Asymptomatic – only apparent on blood test
   b. Uremic Syndromes
      (1) Altered mental status
      (2) Pericardial disease
      (3) Pruritis
      (4) Nausea/vomiting
Presentation of Acute Renal Failure:

3. Metabolic
   a. Hyperkalemia - dysrhythmias
   b. Metabolic acidosis
   c. Hyponatremia
   d. Hypocalcemia
      (1) Usually asymptomatic
      (2) May result in tetany after bicarb given
   e. Hyperphosphatemia
   f. Hypermagnesemia
Presentation of Acute Renal Failure:

4. Hematologic
   a. Anemia – if renal disease is long-standing
   b. Thrombocytopenia
   c. GI bleeding from bleeding diathesis
Presentation of Acute Renal Failure:

5. Active Urinary Sediment
   a. Red cell casts suggest glomerulonephritis
   b. Azotemia in the setting of a normal urinalysis suggests obstruction
Evaluation of the Patient with Acute Renal Failure:

1. **Volume Status!!!!
2. Physical Exam – distended bladder, pelvic tumor/mass, prostate
3. Uremic Syndrome?
4. Exposure to Toxins?
5. Search for pre-renal and post-renal disease – these are typically reversible
6. Laboratory Results
### Test Results

<table>
<thead>
<tr>
<th>Test</th>
<th>Pre-renal</th>
<th>Renal</th>
<th>Acute Tubular Necrosis</th>
<th>Post-renal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine Sodium (mEq/L)</td>
<td>&lt;20</td>
<td></td>
<td>&gt;40</td>
<td></td>
</tr>
<tr>
<td>Fract Excret of sodium (%)*</td>
<td>&lt;1</td>
<td></td>
<td>&gt;2</td>
<td></td>
</tr>
</tbody>
</table>

#### Urine Sodium is Usually Pretty Low!

**Thus, a measurement of a HIGH urine Na+ suggests that the kidneys are “wasting salt”**

<table>
<thead>
<tr>
<th>Kidney size</th>
<th>granular casts</th>
<th>WBC casts</th>
<th>pigmented cellular casts</th>
<th>granular casts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Poor uptake</td>
<td>Good uptake</td>
<td>Variable</td>
<td>Increased</td>
</tr>
<tr>
<td>Radionuclide Study</td>
<td>Delayed excretion</td>
<td>Delayed excretion</td>
<td>No excretion</td>
<td>Good uptake</td>
</tr>
</tbody>
</table>

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*Fractional Excretion of sodium % = (Urine Sodium / Urine Osmolality) / (Serum Sodium / Serum Osmolality)*

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*Serum BUN/Creatinine ratio >20:1 ~ 10:1 suggests that the kidneys are “wasting salt”*
Burton D. Rose, MD
Nephrologist
Boston, MA
“Up to Date”
1998
“Urine sodium concentration — The urine sodium concentration tends to be low in prerenal disease (<20 meq/L) in an appropriate attempt to conserve sodium, and high in ATN (>40 meq/L) due in part to the tubular injury. There is, however, frequent overlap resulting in many cases from variations in water reabsorption which can also affect the urine sodium concentration. As an example, a prerenal patient who is highly water-avid due to increased secretion of antidiuretic hormone may have a higher than expected urine sodium concentration despite excreting relatively little sodium. Conversely, decreased water reabsorption in ATN can lower the urine sodium by dilution. The net effect is that the fractional excretion of sodium (FENa) is a better test, because it evaluates only sodium handling (the fraction of the filtered sodium that is excreted) and is not affected by changes in water reabsorption.”
Management of the Patient with Acute Renal Failure:

**Remember folks!!**

Potassium rises 0.6 mEq/L for every decrease of 0.1 pH on the ABG. So, even if the potassium is 6.4, if the pH is 7.00, then the patient has a corrected K+ of 4.0.

- (1) Calcium
- (2) Bicarb
- (3) Insulin/Dextrose
- (4) Kayexalate (Na polystyrene sulfonate)
- (5) Lasix
- (6) Albuterol
Management of the Patient with Acute Renal Failure:

1. Treat Complications
   b. Volume Overload
   c. Hypocalcemia
      (1) May be precipitated by Bicarb
      (2) Tetany – calcium gluconate
   d. Hypermagnesemia
      (1) Avoid extra magnesium in antacids or laxatives
Management of the Patient with Acute Renal Failure:

1. Treat Complications
   
   e. Hyperphosphatemia
      
      (1) Limit phosphate in diet
      (2) Use phosphate absorbing antacids

   f. Pericarditis
      
      - In ARF, is indication for urgent dialysis

   g. Correct bleeding diathesis/platelet abn.
      
      (1) Cryoprecipitate
      (2) DDAVP (raises Factor VIII four-fold)
Management of the Patient with Acute Renal Failure:

2. Pre-renal – improve blood flow to kidney
   a. Replace volume
   b. Dopamine
   c. Relieve compromised blood flow
      (1) Renal artery stenosis and ACE inhibitor
      (2) Trauma/tumor
Management of the Patient with Acute Renal Failure:

- Hyperkalemia
- Volume Overload, with cardiopulmonary compromise
- Acidosis
- Hypertension, severe and uncontrolled
- Drug toxicity – salicylate, lithium, theophylline (charcoal hemoperfusion preferred), methanol, ethylene glycol

Possible Indications:
Pericarditis, progressive altered mental status from uremia

b. Transfer, if nephrologist or dialysis unavailable
Acute Renal Failure:

Glomerulonephritis

1. Syndrome of hematuria, proteinuria
2. Immune mediated inflam. of glomerulus
3. Post-streptococcal most common in childhood
4. Presentation
   a. Edema
   b. Hematuria
   c. Hypertension
d. ARF
5. Management
   a. Most admitted =>> Definitely consult!
   b. Manage complications of ARF
MANAGING COMPLICATIONS OF ARF

- Hyperkalemia
- Volume Overload
- Hypocalcemia
- Hypermagnesemia
- Hyperphosphatemia
- Pericarditis
- Pre-renal (Replace volume, dopamine, Relieve compromised blood flow)
- Renal
- Post Renal – Relieve obstruction
- Encourage Urine Flow (Mannitol, Lasix)
- Possible need for Dialysis
Acute Renal Failure:
Nephrotic Syndrome

1. Syndrome of proteinuria, hypoalbuminemia, edema, hyperlipidemia
2. Glomerular disease causing protein leaking
3. Peak: Ages 2-7 years – minimal change nephrotic syndrome most common

4. Clinical Presentation
   a. Edema
   b. Hematuria and hypertension unusual

5. Management
   a. Most admitted – definitely consult
   b. Manage complications of ARF
   c. Lasix is diuretic of choice
Chronic Renal Failure

1. Irreversible renal function loss
2. Kidneys usually small and scarred
3. GFR > 25% normally tolerated well
4. GFR < 25% caused end-stage renal disease
5. Evaluation is centered around ruling ARF and ARF superimposed on CRF
6. Treatment
   a. Dialysis
   b. Transplant
Hemodialysis Complications

1. Vascular Access
   a. Fistula is vein redirected, vs. graft is artificial
   b. No blood draw, tourniquet or BP in extremity with shunt
   c. In life-threatening emergency, can access shunt:
      (1) Sterile technique
      (2) DO NOT PUNCTURE BACK WALL
      (3) Will need pump for IV fluid
      (4) Use non-occlusive pressure to halt bleeding
Hemodialysis Complications

1. Vascular Access
   d. Shunt thrombosis – loss of thrill, then consult vascular surgeon: Some are using thrombolytics
   e. Shunt infection
      (1) Red, warm, tender
      (2) IV antibiotics to cover skin flora
      (3) Likely admission
Hemodialysis Complications

2. Bleeding – Platelet dysfunction and/or heparin complications

3. Hypotension – must search for cause
   a. MI
   b. Pericardial Tamponade
      (1) Due to bleeding
      (2) Symptomatic with volume reduction
   c. Sepsis
   d. Bleeding secondary to anticoagulation
   e. Excessive fluid removal
Hemodialysis Complications

4. Hypokalemia
   a. Only occurs during dialysis
   b. Causes ventricular irritability
   c. With Ventricular fibrillation during dialysis, think about hypokalemia
   d. Digitalis toxicity may be precipitated by hypokalemia from dialysis
Hemodialysis Complications

5. Disequilibrium syndrome
   a. Due to rapid change in osmolality
   b. More common during first dialysis session
   c. Nausea, vomiting, headache
   d. Altered mental status, seizure may occur
   e. Treatment – infuse osmotically active substance
   f. Consider other causes of CHS dysfunction, especially in previously stable dialysis patient
The understanding of renal physiology connects us directly to the evolution of the universe.
As with ALL subjects in science, if you don’t give this connection a REGULAR CONSIDERATION...
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?? or !!