Dialysis Options in the ICU: What is the Buzz about CRRT?

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Conflict of Interest??

No
Objectives:

1. Discuss the impact of Acute Kidney Injury (AKI) in the critical care setting
2. Describe the fundamental transport mechanisms for solute removal
3. Compare and contrast the advantages and disadvantages of various dialysis options for AKI including intermittent hemodialysis (IHD), peritoneal dialysis (PD), and continuous renal replacement therapy (CRRT).
Meet The Kidney
- 1,000,000 Nephrons
- Loose 50% before signs and symptoms
- Renal Insufficiency - 75% lost
- End Stage Renal Failure - 90% lost
Renal Blood Flow

- 20%-25% of cardiac output
- 1200 ml/minute
- Decreases with age
- Auto regulation lost when MAP is <70 mm/hg
- Ischemic injury occurs when MAP < 60 mm/hg for > 40 minutes
Functions of the Kidney..

1. Elimination of metabolic waste
2. B/P regulation
3. Erythrocyte production
4. Activation of Vitamin D
5. Auto regulation of blood flow
6. Acid-base balance
7. Fluid /Electrolyte balance
Ricci et al (2007) in a review of 13 studies concluded the mortality w/ diagnosis of AKI was 31.2% compared to 6.9% for patient w/o diagnosis.

- 50% of AKI develops in the hospital
- Development of AKI is associated with
  - 7-fold increase in death
  - 4 fold increase in LOS
  - 4 fold increase in likelihood of transfer to ICU
• Sudden *failure* of renal function either due to decrease perfusion, obstruction, or intrarenal disease, when the glomerular filtration rate decreases, metabolic waste builds up and the body’s internal balance is lost.

• *What is “sudden”? What levels are “in balance”?* Is a Cr of 2 AKI? *What about a decrease in UO? How much of decrease? Failure: will they recover?*
GFR Criteria* | Urine Output Criteria
---|---
**Risk**
Increased SCreat x 1.5 or GFR decrease > 25% | UO < 0.5 ml/kg/h x 6 hr
**Injury**
Increased SCreat x 2 or GFR decrease > 50% | UO < 0.5 ml/kg/h x 12 hr
**Failure**
Increase SCreat x 3, GFR decrease 75% OR SCreat ≥ 4 mg/dl | UO < 0.3 ml/kg/h x 24 hr or Anuria x 12 hrs
Acute rise ≥ 0.5 mg/dl
**Loss**
Persistent ARF** = complete loss of kidney function > 4 weeks
**ESKD**
End Stage Kidney Disease (> 3 months)

Yaklin, 2011
Risk vs. Injury vs. Failure

Figure: Mortality by RIFLE class.
AKI Defined

• An abrupt (within 48 hr) reduction in kidney function currently defined as an absolute increase in serum Creatinine of > 3.0 mg/dl, a % increase in serum Creatinine > 50%, or a reduction in UO < 0.5 ml/kg/hr for > 6 hr.
Risk Factors for AKI

- Increasing age
- Concurrent valve and bypass operations
- Renal dysfunction
- Left ventricular dysfunction
- Prolonged bypass time
- Vasopressors
- Volume depletion
- Amino glycoside therapy (Tobramycin & Gentamycin)
- Radiocontrast exposure
Classifications of AKI

- Post Renal
- Intrinsic Kidney Injury
- Pre Renal
• Decreased renal perfusion
• Intravascular volume depletion
• Cardiac failure
• Ischemia to nephrons
• BUN/Cr  > 10:1
• Urine Sodium  < 20
• Specific Gravity of urine > 1.020

Most common cause of ARF among critically ill patients.
Intra-Renal

- Characterized by destruction of renal parenchyma
  - Prolonged pre-renal → ischemia and cell necrosis
  - Infection agents or toxins
  - *Acute Tubular Necrosis (ATN)*, *Acute Interstitial Nephritis (AIN)*, *Contrast-induced Nephropathy (CIN)*
- May also include glomerular nephritis, renal vascular disease, malignant hypertensive.
- Treatment: Supportive care/RRT
• Prolonged urinary tract obstruction leading to back pressure within the kidneys resulting in drop in the GFR

• Causes include- kidney calculi, blood clots, benign prostatic hypertrophy, malignancies

• Least common
  • *Always irrigate foley with decreased UO

Yaklin, 2011
Acute Kidney Injury

Prerenal (60–70%)
- Tubular cell injury
  - Ischemia and inflammation (sepsis, surgery, hypoperfusion)

Intrinsic (25–40%)
- Acute interstitial nephritis
- Toxins
  - Direct: aminoglycosides, cis-platinum
  - Vasoconstriction: NSAIDs, cyclosporine A, radiocontrast

Postrenal (5–10%)
- Acute glomerulonephritis
Indications for Dialysis: (A, E, I, O, & U)

- A: Acid/Base disturbances
- E: Electrolyte disorders
- I: Intoxications (Lithium, ASA, etc.)
- O: Fluid over load
- U: Uremia
- Sometimes F 😊
KEY CONCEPT

• The Hemo-filter is the key to CRRT therapy
• During therapy, water, electrolytes, and other solutes are removed as the patient’s blood passes through the semi permeable membrane of the Hemo-filter
• Plasma proteins and cellular components are too large to pass through the membrane and thus returned to the patient

Molecular Weights: (Daltons)

Large
- Albumin (~ 60,000)
- Beta2 Microglobulin (11,800)

Middle
- Insulin (5,200)
- Glucose (180)
- Uric Acid (168)
- Creatinine (113)

Small
- Phosphates (80)
- Potassium (35)
- Phosphorus (31)
- Sodium (23)
Molecular Transport

- Diffusion
- Convection
- Adsorption

**type of needed transport dependent upon size of molecule**
Diffusion/Dialysis

• Movement of solutes from an area of high concentration to an area of lower concentration

• Diffusion rate dependent upon the concentration, size, & electrical charge of the solutes

• The concentration gradient is maintained by running the dialysate countercurrent to the blood flow

• Small molecules (urea) move easily, while larger molecules remain within the plasma
Diffusion/Dialysis

- Good for small solute removal (<500 Da)
- Diffusion inversely proportional to MW
- Not Removing fluid from blood
  - less hemo-concentration
  (decreases in filter clotting)
Basics of RRT

- **Diffusion**
  - random motion
  - molecule strikes pore

- **Variables**
  - molecular weight (removes small molecular wt. solutes)
    - speed
    - size
  - membrane resistance
  - unstirred layer

Equilibrium
Diffusion: In action

Granny's $K^+$ 8.0

Dialysate

$\frac{K}{4.0}$
Granny’s Bicarb: 6

How high will her Bicarb go?
Granny’s Bicarb 32

How Low will her bicarb go?

Dialysate

Bicarb 22
Basics of RRT

- **Ultrafiltration**
  - hydrostatic/osmotic
  - pressure gradient drives $\text{H}_2\text{O}$ across membrane
  - with osmotic UF there is an osmotic driving force

![Diagram of Ultrafiltration](image)
Convection

- Pressure gradient is set up across the filter (semi-permeable membrane)
- Water is pushed across the membrane and carries dissolved solutes with it → “SOLUTE DRAG”
- In order to effectively remove molecules by convection alone, typically at least 1L/hr must be pulled through the membrane (for most adults)
  - 24 liters/day
• Convection
  • solutes swept along with water
  • removes middle molecular wt solutes
  • must be small enough to fit through pore
  • the more UF you have the more solutes you clear
Convection

- Removes plasma water as it seeps through the membrane (water being removed = ultra filtrate)
- *membrane permeability is KEY to process
- Particles <15,000 Da can potentially be removed
- Produces hemo-concentration
  - Hct prefilter ~ 40%
  - Hct Post-filter ~ 45%
Convection vs. Ultrafiltration

- **Ultrafiltration**: Movement of FLUID through a membrane caused by a pressure gradient.
- **Convection**: Movement of SOLUTES with water flow across a semi-permeable membrane. (solute drag)
- **convection → solvent (water) is pushed across the membrane (a process called ultrafiltration) in response to the trans-membrane pressure gradient.**
Hemodialysis
Hemodiafiltration

Blood In (from patient)

Blood Out (to patient)

LOW PRESS → HIGH PRESS
LOW CONC → HIGH CONC

Dialysate Solution

Repl. Solution

to waste
RRT MODALITIES

Intermittent
- IHD
- SLED

Continuous
- CRRT
  - SCUF
  - CVVH
  - CVVHD
  - CVVHDF
- PD
1. PD: Peritoneal Dialysis
2. IHD: Intermittent Hemodialysis
3. SLED: Sustained Low Efficiency Dialysis
4. CRRT: Continuous Renal Replacement Therapy
Peritoneal Dialysis
Peritoneal Dialysis

- Uses the peritoneum as the dialyzing semi permeable membrane
- Slow and continuous
- Produces an over distended ABD \(\rightarrow\) compromise pulmonary function.
- Does require a sterile intact peritoneum
  - Often not present with sepsis
  - Surgical sites
• Peritoneal dialysate baths → hyperglycemic

• Dialysate baths have Lactate as base
  • Lactate is converted in the liver to bicarb-good thing
  • If lactate levels are high > 2
    • A sign of global tissue hypo-perfusion or just a consequence of the dialysis bath ???
IHD: Advantages

- IHD: widely available, good solute control.
- Excellent solute control with daily therapy
IHD: Disadvantages

• Nutritional and Fluid Restrictions
• Disequilibrium Syndrome: Rapid decline of urea and Na leads to shifts of extracellular water to intracellular space. (cerebral edema)
• Contraindicated in patients with acute liver failure or in presents of cerebral edema
• Decreased perfusion $\Rightarrow$ further renal damage
IHD: Disadvantages

- Poorly tolerated by hemodynamically unstable patients
- Rapid reduction in blood volume leads to intravascular volume depletion
- Use of Arterial Access
  - bleeding
  - infection
  - clotting
  * Limited amount solute removal limits therapies
• Korean War Lessons

• Highly effective in removing water and solutes

• Inability to manage hourly fluctuations in volume status
SLED: Sustained Low-Efficiency Dialysis

Uses conventional hemodialysis: run slower with blood pump speeds and low dialysate flow rates

- Combines the best of both CRRT/IHD
  - Improved Hemodynamic Stability (slower rates)
  - High Solute clearance without expense of CRRT
  - Can be done on Intermittent bases (6-12 hrs)
  - Work around tests/procedures
Introduction to CRRT

• CRRT = Continuous Renal Replacement Therapy

• Defined as:

   “Any extracorporeal blood purification therapy intended to substitute for impaired renal function over an extended period of time and applied for or aimed at being supplied for 24 hours/day.”*

• CRRT: Continuous Renal Replacement Therapy

• 4 Types of Therapy:
  1. SCUF
  2. CVVH
  3. CVVHD
  4. CVVHDF
<table>
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<th>CRRT:</th>
<th>Hemodialysis:</th>
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<tbody>
<tr>
<td>1. Continuous</td>
<td>1. Intermittent</td>
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<tr>
<td>2. Can utilize venous access</td>
<td>2. May use arterial access</td>
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<td>3. Less hemodynamic instability</td>
<td>3. Greater hemodynamic instability</td>
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<td>4. Constant adjustment of fluids</td>
<td>4. Faster clearance related to greater flow</td>
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<td>5. Increased clearance of</td>
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<td>r/t convective mode of solute</td>
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WHY CRRT??

• CRRT provides continuous therapy
• It closely mimics the body’s natural renal function
  • Slow/gentle $\rightarrow$ well tolerated by hypotensive patients
  • Removes targeted amounts of fluid and waste
  • Avoid peaks and troughs assoc. with IHD
  • Unlimited NTR, Fluid, Blood Products (no need for fluid restriction)
Advantages of CRRT

• Minimal changes in plasma osmolality

• Better control of azotemia, electrolytes, and acid/base status

• Easier administration of parenteral nutrition and medications
Advantages of CRRT

• Effective removal of fluid

• Removal of large molecules with cardio depressant, vasodilatory, and inflammatory properties*

• Removal of toxins with larger volumes of distribution
CRRT: Immune Modulation Therapy

- Cytokines: low MW, soluble protein messengers produced in response to an antigen.
- Cytokines bind to cytokine specific receptors on other cells of the immune system and influencing the function of the immune system.
Jury is Still Out...

• Cytokines are readily absorbed into the filter membrane.

• Some cytokines are removed by CVVH.

• However, there are no studies demonstrating these effects to be clinically significant.

Disadvantages of CRRT

- Specialty education required/labor intensive
- Frequent clotting of filter and access
- Need for anticoagulation
- Increased pt. acuity
- Decreased pt. mobility
- Drug dosing based on 24/hr. run....how often does your CRRT run for 24/hrs.?
Indications for CRRT
A, E, I, O, U

- **Hemodynamic instability**
- Literature is mixed at best for HD vs. CRRT
  *Anecdotal support based on premise of cytokine removal by CRRT.*
  Strong double blinded studies do not support superiority of CRRT to HD for:
  - Severe Sepsis
  - ARDS
  - Rhabdomyolysis
  - Tumor lysis (if HD unable to match solute release into the systemic circulation)
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